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The
Medical Clinics of Chicago

VOLUME 2 NUMBER 1

CLINIC OF DR. ARTHUR R. EDWARDS

MICHAEL REESE HOSPITAL

THE USE OF DIGITALIS

General Indications and Contra-indications. Digitalis Indicated for Cardiac Insufficiency of Whatever Type. Action in Individual Valvular Lesions.

Toxic Manifestations. Avoidance of Cumulative Action.

Preparations and Administration. Adjuvants and Symptomatic Therapy.

IN the various examples of stasis or cardiac insufficiency which you have seen clinically you may observe that the essential symptomatology pivots on the myocardium. Conventionally, we classify heart diseases as those of the pericardium, myocardium, and endocardium—besides those of nervous disturbance. Practically, from the therapeutic standpoint, most cardiac affections are, on last analysis, myocardial. Valvular diseases, sharply distinguished from each other by their peculiar physical findings, attract attention first when the overstrained heart muscle becomes more or less "incompetent." The fresh effusion of pericarditis causes other than local signs and symptoms chiefly when the fluid presses upon the heart and obstructs its complete diastole, and the later adhesions impede the systole by anchoring the heart to the chest wall and mediastinum. The brunt of the chronic intoxications and the acute infections is sustained by the heart muscle. The arterial cardiopathies, as myofibrosis or angina pectoris, inflict injury upon the myocardium

by interfering with the access of blood to the heart muscle; fatty heart, the senile heart, heart-block, and many other arrhythmias are simply failing competence of this contractile muscle.

The heart possesses not only a certain *reserve power*, but in favorable instances its musculature *hypertrophies*. Every hypertrophy is more or less temporary in its compensation, and ultimate cardiac failure is inevitable, either temporarily, "disturbed compensation," or permanently, "loss or rupture of compensation," the asystolie of French writers. Failing cardiac competency is attended by (1) changes in the heart itself and altered movement of the blood; (2) symptoms in organs remote from the heart, as the congested liver, etc.; and (3) reflex symptoms, as hyperalgesia over the heart or liver. The general tendency in stasis is to reduce arterial tension, increase venous pressure, retard the capillary flow, and promote embolism. The blood stagnating in the veins may cause capillary resistance and, therefore, raise arterial pressure, even though the circulation is embarrassed; in stasis, also, the cyanosis may raise arterial tension from poor aëration of the blood. Mackenzie maintains that heart failure may develop without fall in arterial pressure, but here there is no loss of tonicity; when the heart becomes atonic (dilated) the blood-pressure falls, to rise again when tonicity is restored. Dilatation of the compensatory type appears first and incites hypertrophy; as hypertrophy reaches its limit, the dilatation of the second type appears—the dilatation causing stasis. Circulatory disturbances may be spontaneous or induced by psychical excitement, physical exertion, intercurrent disease, recurrent endocarditis, malnutrition, or excessive use of alcohol or tobacco.

Broken compensation may be acute, gradual, or terminal, as by coronary occlusion, and is evidenced at first by symptoms not necessarily cardiac, as fatigue, vague thoracic or abdominal pain or hyperesthesia, so-called neurasthenic symptoms, inertia, heaviness of the limbs, etc. The greatest therapeutic success follows the recognition of these extracardial evidences, not awaiting the severer signs of stasis—*e. g.*, dyspnea, irregular pulse, and dilatation.

Digitalis was first recommended by Wm. Withering (1775) and its physiologic action demonstrated by Traube. Digitalis attaches itself to the heart muscle, and, becoming fixed in this tissue, it is slowly given off and eliminated. Operating on the myocardium, vagus, vasomotors, and other muscular tissue it aids the heart to elicit and co-ordinate all its latent power, but the drug cannot augment the absolute power of the heart.

General Indications.—(*a*) *Dilatation; digitalis restores tonicity* to the heart muscle upon which the drug exercises its main influence, *i. e.*, it overcomes dilatation. (*b*) *Irregularity*, or functional dissociation, is often corrected by digitalis; it stimulates the function "rhythmicity" by action on the heart muscle, the ganglia, and the peripheral filaments of the vagus. Some claim that the drug acts chiefly on the vagus, and through it operates more effectually and more primarily upon the auricles than ventricles, which latter are impressed secondarily by way of the auricles and bundle of His. In about 80 per cent. of patients helped by digitalis there is continuous irregularity due to auricular fibrillation (Mackenzie); in this condition the auricles contract rapidly and disorderly and convey impulses to the ventricles more rapidly than normal; digitalis regulates by decreasing conduction through the bundle of His, thereby protecting the ventricles from undue stimulation from the auricles. Digitalis is naturally more efficacious in recent than in ancient cases of auricular fibrillation. Digitalis does not help extrasystoles. (*c*) *Rapid, weak heart action:* Digitalis slows the heart by action on the vagus. It tends to abolish abortive cardiac contractions and synergizes the systole of the chambers (increased contractility), as is shown by the strong, almost tetanic, systolic spasm of the heart in cases of digitalis-poisoning. The slower and stronger systole empties the heart chambers more completely, forces the venous and arterial blood into the main arteries, and squeezes the venous blood out of the heart muscle itself, thereby improving the intrinsic circulation of the heart. The slower, more complete diastole allows better filling of the heart and drainage of the engorged veins. In stasis, the heart, lungs, liver, kidneys, etc., show arterial ischemia, venous hyperemia, and fatty change in the

vessel walls, with its pathologic sequences—vessel rupture as in the hemoptysis of brown induration of the lungs, fatty degeneration as in the mottled nutmeg liver, and, in extreme cases, fibrosis, as myofibrosis cardis or cyanotic induration of the kidneys and liver. Except in irreparable cases all these pathologic changes, in the heart itself as well as in more distant organs, are improved or removed by digitalis.

Digitalis was formerly described as raising the arterial tension by increasing the working power of the heart and by its centric and local action on the vasomotors; often, however, arterial pressure is not raised, and then digitalis probably equalizes the distribution of the blood and lessens tension due to edema and poor aëration of the blood. The most marked vasoconstriction is in the splanchnic area. As noted above, heart failure without loss of tonicity may be observed without a fall in arterial tension; heart failure with loss of tonicity (dilatation) may be associated with low blood-pressure, which latter may rise to normal, when rest or digitalis restores tonicity and thus removes the dilatation. On the other hand, stasis may coexist with blood-pressure higher than normal, which is explained by the cyanosis (CO_2 narcosis) or by increased capillary resistance offered by stasis, anasarca, etc.

Digitalis, in other words, may remove stasis (*e. g.*, anasarca, ascites) without raising the blood-pressure, or, indeed, even with a lowering of blood-pressure. Fränkel has shown that the systolic pressure in a healthy individual is not elevated by digitalis, but remains constant. This may be explained thus: If an animal is given atropin previous to the administration of digitalis, the pulse is not slowed by the latter, but the blood-pressure is markedly increased; consequently, it must be that the slowing of the pulse-rate opposes the action upon the blood-pressure. Likewise in man, if the slowing of the pulse is avoided, the blood-pressure will be increased by the digitalis. This may be done by injecting hypodermically $\frac{1}{100}$ grain of atropin. On the contrary, it is shown that the acceleration of the pulse caused by atropin does not affect the blood-pressure in the healthy subject. In short, the slowing of the heart prevents a rise of blood-pressure.

It is seen then that digitalis increases tonicity, rhythmicity, and contractility, while lessening excitability and conductivity. All in all, aside from details, *digitalis is indicated for cardiac insufficiency of whatsoever type*, which fact must be kept in view when we consider its contra-indications.

In the individual valvular lesions digitalis acts as follows: (*a*) In *mitral stenosis* the longer diastole drains the left auricle and lung vessels, and the stronger systole forces the retarded blood through the stenotic orifice; particularly in this lesion auricular fibrillation arises; (*b*) in *mitral insufficiency* the best results are obtained, the leaking cusps are better coapted, and the stronger systole fills the aorta and pulmonary artery; (*c*) in *aortic stenosis*, when dilatation occurs or the hypertrophy is inadequate, digitalis is valuable; (*d*) in *aortic regurgitation* it is theoretically contra-indicated, since it increases the diastole and hence the leakage; it is, however, as valuable in aortic decompensation as in mitral.

Digitalis is *contra-indicated* (*a*) in balanced compensation; (*b*) when the vessels are fragile, as in aneurysm, and in advanced atheroma on account of the risk of brain hemorrhage; (*c*) in fatty degeneration of the heart, except when it results from stasis and ischemic malnutrition; (*d*) in vascular contraction. In this last instance it is combined with nitroglycerin, strophanthus, or potassium iodid to "unlock the arteries." The drug is not absolutely contra-indicated in the high tension of arteriosclerosis and nephritis, for, paradoxic as this may seem, it may relieve by lessening dyspnea and carbon-dioxid narcosis and by equalizing the circulation. (*e*) Of late irregularity of the heart is considered a contra-indication, especially by Mackenzie, who stops digitalis as soon as irregularity develops. In partial heart-block digitalis may work harm; it may prove beneficial in some cases of complete heart-block. Mackenzie discontinues digitalis in severe cases of mitral stenosis when the presystolic murmur disappears or when systolic waves appear in the jugulars, for then some heart-block has developed. Extrasystole is also a contra-indication. (*f*) Idiosyncrasy; individuals react differently to the drug, some exhibiting hypersensitiveness (*v. i.*, toxic symptoms). (*g*) As Janeway pertinently remarks, digitalis is

indicated only in ventricular weakness and not in paroxysmal tachycardia, nephritic edema, inflammatory effusions, or palpitation. (*h*) Digitalis may fail to operate unless a restricted diet, rest, free purgation, venesection, and mechanical removal of the anasarca have been first employed. Briefly, as to these items, the *Karell diet*, 4 to 6 ounces of milk every four hours, is a low calory diet; its effects are frequently remarkable in dyspnea; it is a "protective diet," imposing less work on the liver and gastro-intestinal tract, reducing the fluids ingested, decreasing alimentary intoxication, and sparing the kidneys in the matter of eliminating salt and protein. *Rest in bed* accelerates the action of digitalis and is desirable when possible; it is, however, a folly, cruelty, and impossibility to enforce the dorsal decubitus in severely dyspneic patients; these unfortunates should be allowed to sit in a chair, with side-supports for the head. *Free purgation* depletes the portal circulation and thus relieves the heart; constipation increases dilatation. Often digitalis is nearly inert until *venesection* is performed, which relieves the dyspnea, cyanosis, dilatation, and tricuspid leakage; venesection raises a low blood-pressure by relieving the above-mentioned symptoms; if an increased blood-pressure, due to a carbonic dioxid excess is present, it is lowered by phlebotomy. The relief afforded the circulation by incision of dropsical members or paracentesis will be considered later under another caption. The Leipzig school exhibits digitalis on the first sign of decompensation.

Many of these contra-indications are relative only, since digitalis is indicated by cardiac insufficiency of any of the five types of Sahli (cardiac, pulmonary, high-pressure stasis, vasomotor, or splanchnic insufficiency); it may be tested out, then, in decompensation due to acute endocarditis (where experimental evidence favors its use), chronic myocarditis, arteriosclerosis, hypertension, coronary disease, tachycardia, etc.

Toxic Manifestations.—Gastro-intestinal symptoms are generally from stasis; sometimes vomiting is due to rejection of the drug by a disordered stomach or, several hours later, to toxic irritation of the stomach or the medulla by the absorbed drug; decomposition of the digitalis (*e. g.*, in the form of the infusion) may

initiate nausea; there may be diarrhea with green stools. Digitalis causes contraction of all unstriped muscular tissue (gastric, vascular, muscular). It may cause delirium, headache, diplopia, blindness to green colors, blueness of the scleræ, exophthalmos, precordial oppression, palpitation, extreme weakness, pallor, renal suppression, violent cardiac overaction, or convulsions (decomposition of the drug).

Cumulative action may attend the use of any efficient preparation, since the heart never becomes anesthetic to digitalis; digitalis preparations without cumulative action are inert. It is averted (*a*) by intermittent administration, although some patients tolerate digitalis continuously for months or years, as Thomeyer's patient, who in eight and a half years took over 15 ounces of pure digitalis; (*b*) by great care in ambulatory cases; (*c*) by care in cardiac dropsy, on relief of which toxic quantities may be resorbed from the tissues, precisely as uremia may ensue on resorption of anasarca in renal insufficiency; (*d*) by care in intercurrent febrile affections, for toxic effects often follow the fall of temperature; toxemia considerably neutralizes the slowing action of the vagus; and (*e*) by withdrawal on the appearance of vomiting, syncope, reduced urine, or irregular or very slow pulse, a sign of danger; here atropin may be advantageously combined with the digitalis.

Preparations and Administration.—Among the standardized official preparations the powdered leaves stand first; the powder deteriorates little, even after eight years, and is given in doses of 1 to 1½ grains. The tincture (10 minims) deteriorates, but much less than the infusion (1 to 2 drams). For hypodermic use, the fluidextract (2 minims), well diluted, is least irritating. Beginning with small doses of digitalis, to avoid sudden overtaxing of the heart, the dosage is increased. Withering, who discovered digitalis, pushed the drug until it acted on the pulse, kidneys, stomach, or bowels, etc., its effects appearing in two to three days.

The infusion must be freshly prepared, as it is prone to decompose and, therefore, to cause gastric irritation; some clinicians would dispense with this preparation because of this tendency. It is excellent for rectal use when the stomach is irritable. Re-

garding the active principles there is the greatest confusion. The *tincture* is thought to be the best cardiant because it contains digitalin and digitoxin. The writer has found digitalin almost inert. *Digitoxin*, in the author's hands, has often strengthened the weak and irregular heart when digitalis has failed ($\frac{1}{400}$ grain three times a day); it is the most potent principle of digitalis, but it easily disorders the stomach, and often cannot be given for more than six or eight doses. It is often combined most advantageously with champagne. Fränkel fears the preparation because he finds such a narrow margin between its therapeutic and its toxic dose.

Cloetta's *digalen* is a soluble non-irritant digitoxin and may be given intravenously, 1 c.c. equalling 0.3 mg., or $\frac{1}{180}$ grain. The preparation acts promptly, though it is somewhat irritating when given hypodermically. Cumulative effects are least likely when the kidneys are intact. Some pharmacologists decry the value of digalen. My personal experience with it has been most satisfactory. *Digipuratum* is an active purified product from which is eliminated the digitonin, the element irritating the stomach and intestines; the removal of the saponins make it a fat-free product. Gottlieb contends that the absorption of digitoxin, the most active constituent of digitalis, is more rapid from digipuratum than from the infusion or tincture; it is slower when great portal stasis exists. The advantage of greater stability and more accurate dosage is claimed; it is given in powder or tablet form (0.1 gram—$1\frac{1}{2}$ grains) every twelve hours, or as a fluid in sterilized tubes for emergency intravenous use. Rapid absorption, lack of alimentary irritation, and free diuresis are claimed for it. Like digalen and digitalone, it is expensive.

Of *substitutes* for digitalis it can scarcely be maintained that there are any because other cardiants are distinctly inferior. (*a*) *Strophanthus*, 5 to 10 minims of the tincture every eight hours, may be given. Advantages: It is more rapid in action than digitalis, perpetuates the effects of digitalis, and is indicated in the irregular heart of mitral disease. It is used to advantage in children under twelve years, in cases where we are waiting for the digitalis to act, and in dilatation where the blood-pressure is

high. Disadvantages: It is less certain and less energetic; it is inferior in edema and does not give tone to the vessels.

The tincture deteriorates rapidly in dilution or aqueous compounds. Of late, *strophanthin*, $\frac{1}{60}$ grain once daily, has been recommended for intravenous administration. It is said to be rapid in action and efficient, but its therapeutic dose and toxic dose are not far apart and it is inferior to digitalis. It must be used with caution in nephritis, high arterial pressure, and bradycardia. Its toxic symptoms are those of digitalis-poisoning, including irregularity of the pulse, partial heart-block, vomiting, etc. It is eliminated by the bowel. It should not be used if digitalis has been recently employed. (*b*) *Strychnin* stimulates the vagus, vasomotors, and the nervous, gastric, and blood-making tissues. Given carelessly, it may irritate the heart, decrease the urine by spasm of the renal vessels, or even induce rupture of the vessels. (*c*) *Spartein sulphate* is given in capsule form because of its bitterness ($\frac{1}{4}$ to $\frac{1}{2}$ grain every eight hours); larger doses may be given (1 to $1\frac{1}{2}$ grains); it seems also to quiet the nervous system, but is inferior to digitalis. It does not contract the vessels.

Adjuvants and Symptomatic Therapy.—(*a*) *Dropsy.*—Saline or hydragogue *catharsis* often relieves moderate edema. Digitalis is often combined with calomel or blue mass and squills (Guy's pill, also known as Addison's or Niemeyer's).

℞. Digitalis . . . . . gr. x;
Massæ hydrargyri . . . . . gr. vj;
Scillæ . . . . . gr. x.—M.
Ft. capsulas x.
Sig.—One after meals for one or two days.

Mercurials must be used with the greatest care. The author saw, in consultation, a case complicated by nephritis in which the tongue sloughed off after 5 grains of calomel had been given. Desperate stomatitis may result in alcoholic, myocarditic, or nephritic subjects. Calomel acts by its conversion into bichlorid, which often incites a bichlorid nephritis. In isolated luetic aortic leakage mercurials may prove specific. Excess of digitalis is held to suppress the urine by inducing renal spasm. It is better to use

potassium salts, as potassium acetate, 30 grains three times a day, which are less abundant in the tissues than the sodium salts. Diuresis following digitalis is associated with increase of sodium chlorid elimination; restriction of salt has few benefits. *Caffein* with digitalis is beneficial in dropsy when the pulse is slow, the mind dull, and the digestion sluggish; anginoid pains are often relieved by it; it may cause nervousness or insomnia (therefore given early in the day), irregular pulse (therefore combined with rum or brandy), or biliousness; 5 grains should be given, but not for more than three days.

℞. Caffeinæ citratæ..... ℥ss;
Sodii benzoatis..... ℥ss;
Aquæ..... ℥iij.—M.
Sig.—One hypodermicful (½ dram) as indicated.

Caffein stimulates the bulbar vasomotor center and also the peripheral vasomotor system, whereby some elevation of arterial pressure is accomplished; it also acts as a direct renal stimulant. In shock and collapse it surpasses strychnin and camphor (Rhomberg). Caffein remarkably stimulates mental and motor efficiency. *Diuretin*, theobromin sodium salicylate (1 to 1½ drams daily), may act marvelously or not at all, operating on the renal cells directly and possibly also on the heart and vessels. Theocin sodium acetate is easily soluble in water, and in doses of 3 grains may act when others of this type fail. *Canadian hemp* (15 minims fluidextractum apocyni) is hard to obtain pure, but sometimes justifies Benjamin Rush's term, "a vegetable trocar." *Sweats* and the use of pilocarpin are both dangerous and injurious. Mechanical drainage is recommended by scarification, by multiple knife or pinpricks, or by Southey's capillary tubes; but the best method is by a single long *deep incision* down to the bone, by which a quart to a gallon of fluid seeps away in a day (and up to 40 grams of albumin). Ascites from heart disease alone rarely necessitates puncture. The latter is indicated chiefly in coincident liver cirrhosis or chronic peritonitis. Hydrothorax is far more important and thoracocentesis should not be delayed when dyspnea is urgent.

(*b*) *Anemia.*—Digitalis with iron and arsenic often produces remarkable improvement:

℞. Digitalis..... gr. xx;
Ferri reducti..... ʒss;
Arseni trioxidi..... gr. ss.—M.
Ft. pilulæ xx.
Sig.—One pill after meals; take for a week only.

(*c*) *Insomnia.*—It is often difficult to relieve insomnia or unrestful sleep broken by nightmare or sudden "heart starts." Spiritus chloroformi, spts. camphoræ, and spts. etheris compos., *aa*, ½ dram, are often helpful. Sulphonal, 20 grains in hot milk at bedtime, may be tried, but is somewhat dangerous in stasis when used more than three nights in succession. Chloral hydrate is excellent in high tension. In morphin we place our chief reliance.

(*d*) *Dyspnea.*—The ice-bag, chloral, and spiritus glycerylis nitratis, 1 minim, are of value for high tension. *Morphin* may be given in the early stages without hesitation, and especially at night for restlessness and dyspnea; it is excellent while waiting for digitalis to act. It is almost specific and lessens the irritability of the respiratory center. Heroin hydrochlorid ($\frac{1}{12}$ to $\frac{1}{8}$ grain) operates similarly, but in some instances apparently it can be used longer than morphin. Opiates require care in weak heart or Cheyne-Stokes' breathing. Dyspnea is often renal or due to right-sided hydrothorax, chronic bronchitis, or excessive dilatation, when the appropriate treatment is catharsis, paracentesis, potassium iodid, or digitalis, respectively.

(*e*) *Respiratory Symptoms.*—Cough, cyanosis, pulmonary edema, and hemoptysis are usually cardiac symptoms (pulmonary engorgement or brown induration) and are relieved by the cardiants:

℞. Ammonii carbonatis..... ʒj;
Tr. hyoscyami..... ʒiv;
Potassii iodidi..... ʒj;
Tr. digitalis..... ʒj;
Infusi calumbæ.....q. s. ad ʒvj.—M.
Sig.—One teaspoonful every four hours.

Expectorants usually nauseate. Hemoptysis is often beneficial, being a species of lung venesection and is seldom urgent or fatal. Pulmonary edema necessitates phlebotomy, a 30-grain dose of lead acetate, gr. $\frac{1}{100}$ of nitroglycerin, and $\frac{1}{4}$ grain of morphin.

(*f*) *Palpitation and Pain.*—For palpitation and throbbing the ice-bag is the best calmative; a precordial blister or a few minims of tincture of belladonnæ and tincture aconiti are often helpful, with morphin, if necessary.

℞. Fluidextracti belladonnæ..... gtt. xv;
Tr. digitalis..... ℥j;
Aq. laurocerasi.....q. s. ad ℥j.—M.
Sig.—One teaspoonful two or three times daily.

A light evening meal and laxatives are helpful. Iodid of potassium and nitroglycerin relieve the pain, weakness, pallor, or headache of aortic lesions.

(*g*) *Gastric Symptoms.*—These are ominous when the liver is enlarged and there is not much edema. They are due to portal stasis more often than to digitalis, which should then be given by rectum. Phenol, $\frac{1}{4}$ grain every half-hour for five doses, and rectal nourishment are indicated.

CLINIC OF DR. FREDERICK TICE

COOK COUNTY HOSPITAL

SOME CASES OF DIABETES MELLITUS WITH COMPLICATIONS

Gangrene and Pulmonary Tuberculosis Frequent Complications of Diabetes. Relatively High Percentage of Diabetics Die of Acidosis.

March 6, 1916.

CASE I.—DIABETES MELLITUS ASSOCIATED WITH CHRONIC INTERSTITIAL NEPHRITIS, ARTERIOSCLEROSIS, CHRONIC PERIPHERAL NEURITIS, AND A BEGINNING GANGRENE OF THE TOES.

History.—*Present History.*—This patient entered the hospital on the 1st of February. She is sixty-nine years old, and the history obtained at the time of entrance was as follows: Her illness started about one year ago with a marked increase in thirst and the passage of large quantities of urine. The patient also noticed an increase of appetite, with a steadily progressive loss in weight and marked weakness. This condition constantly grew worse and became associated with an itching of the vulva, pain in the legs, a feeling of numbness in the feet, and inability to walk except with a staggering gait. Three months ago the patient came into the hospital and remained under observation for five weeks, with distinct improvement. She went home, but was forced to return on account of the increased thirst and frequency of urination. During her stay at home there was a loss of weight, marked weakness, and a return of the pains in the limbs.

Past History.—She suffered from the usual diseases of childhood. She had an attack of pneumonia twenty years ago; typhoid fever four years ago. She underwent an operation for a uterine tumor six years ago.

Family History.—Her father and mother both died of old age. Four sisters are living and well.

Marital History.—She has been married twice; both husbands were accidentally killed. Of six children, all by the first husband, only one is living, but according to the history the cause of their death could not be determined. There is also a history of one miscarriage. She went through the climacteric twenty years ago.

Habits.—There is no history of the use of alcohol. She drinks tea and coffee. Venereal history is denied.

Physical Examination.—The history has referred to a loss in weight, and our general inspection reveals a definite laxity in the abdominal muscles, a flabby condition of the skin and muscles of the extremities, with little or no tonus. You will also observe the scar from the recent vaccination. This patient was previously vaccinated forty years ago. Examination of the chest reveals nothing of particular importance. Expansion of the two sides is equal. On percussion, there is a slightly hyperresonant note over the entire anterior surface of the chest. The cardiac impulse is rather diffuse; there is no definite apex-beat. The left border of the cardiac dulness is at least one finger's breadth outside of the midclavicular line; the right border is at the midsternum and the base of the heart is at the lower border of the third rib. On auscultation over the lungs anteriorly the breath sounds are perhaps slightly roughened, a little prolonged, and there is an occasional inspiratory râle. Over the heart there is a slight irregularity, but no murmurs. The peripheral vessels are distinctly hard, thickened, and sclerotic—not beaded, but definitely hard.

Percussion of the chest posteriorly gives a slightly hyperresonant note, with the border of the lungs somewhat lower than the normal position—more than a hand's breadth below the lower angle of the scapula. On auscultation, the findings are the same as obtained anteriorly. The breath sounds are slightly roughened, prolonged, and a few râles are heard.

The abdominal muscles are very lax; there is no tenderness on palpation. The lower border of the liver is palpable three fingers below the costal border; the spleen is not palpable. A distended colon can be felt, there having been no stool this morning.

On the right side of the chest posteriorly you can observe an old pigmented scar which the patient says was "a big boil" which "came by itself." She says it broke spontaneously and ran for about three months. That occurred three years ago. On the right leg there is the scar of another sore which she says also came of its own accord a year ago last winter, in 1914. As you can observe, this scar is elliptic, arrow-shaped in outline, and there is a definite increase of pigment around the border while the central portion is relatively white. It is not depressed, but is more delicate than the surrounding pigmented portion.

You will recall from the history that the patient has complained of pains in her limbs, which she says have all been below the knees. We have tried repeatedly to obtain a patellar and tendo achillis reflex, using reinforcement and distracting her attention, but we have been unable to do so. The history informs us that this patient is able to get about, but that she staggers. You will observe also the condition of the middle toe on the left foot. It is hyperemic and especially painful, she tells us, although all of her toes hurt.

The Urinalysis.—The urine was examined the day she entered the hospital. A single specimen taken on that day showed a specific gravity of 1020, alkaline reaction; albumin was found and sugar was present by the Haines test. Subsequent daily examinations have been made for a considerable period of time. From February 1st, the day she entered the hospital, until the 21st sugar was constantly present in the urine in amounts varying from just a mere trace to 2 per cent. by the Purdy method. She was sugar free from the 21st to the 28th; then sugar reappeared in the amount of ½ of 1 per cent. and continued to the 4th of March, when she again became sugar free.

Of course, you cannot fully appreciate the significance of that unless you know something about her diet, especially the amount of carbohydrates.

When this patient entered the hospital she was placed upon a rather rigid diet. At certain intervals she underwent what are known as "starvation days," but in spite of close attention to the

diet she did not become sugar free, but continued to eliminate sugar until February 21st. Then she was sugar free until the

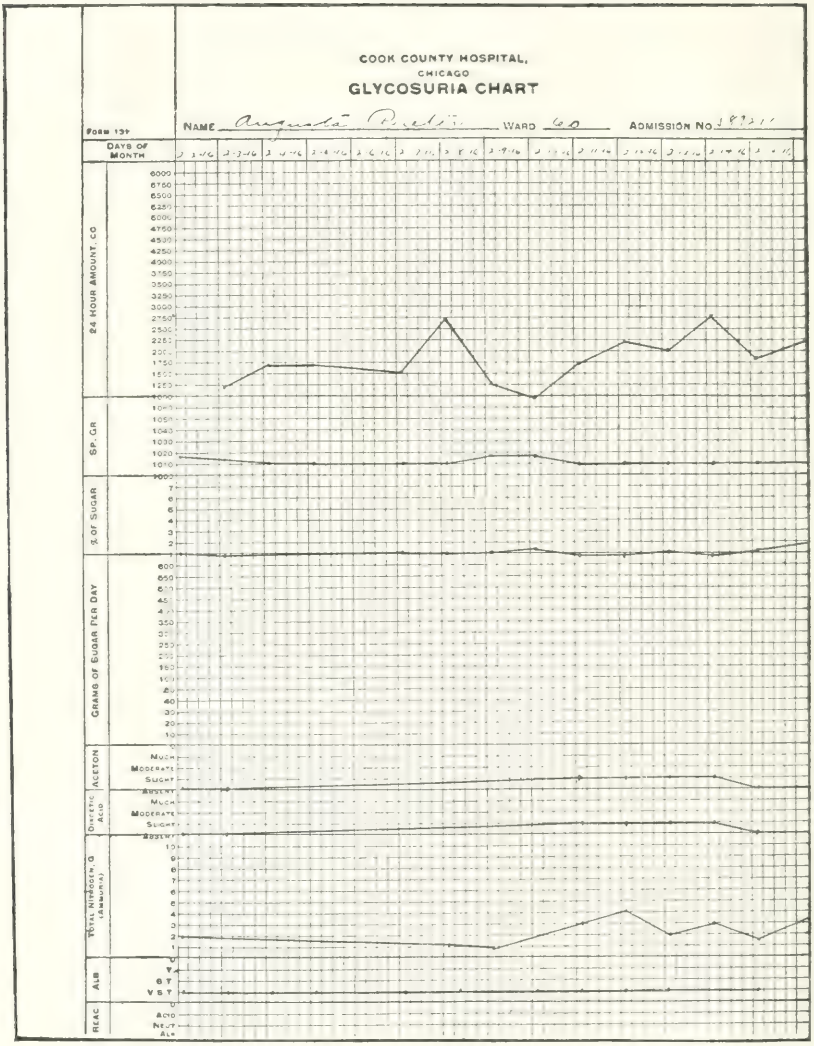

Fig. 1.

28th, when sugar reappeared and was present until March 4th, on exactly the same diet.

Further examination of the urine revealed the constant presence of albumin varying from a mere trace to 3 grams.

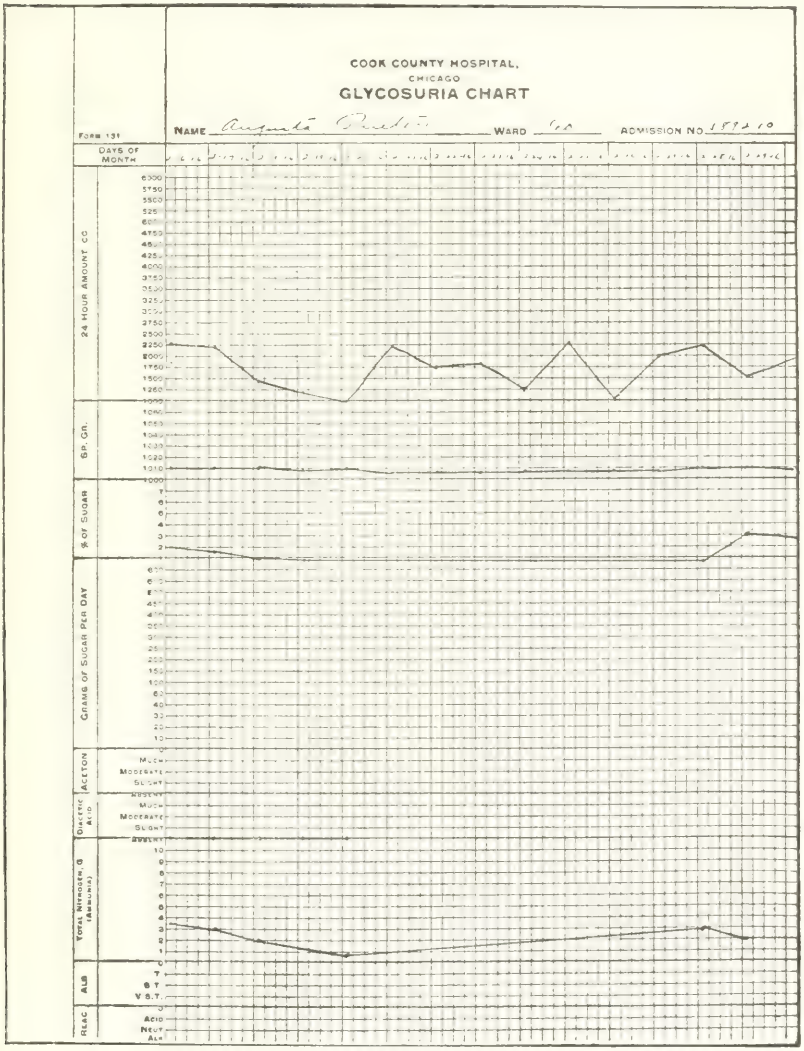

Fig. 2.

Acetone and diacetic acid have been quite constantly present. The ammonia determination, as recorded almost daily, has

varied from 0.56 to 2.01 grams in twenty-four hours. Hyaline and granular casts have been present in every specimen.

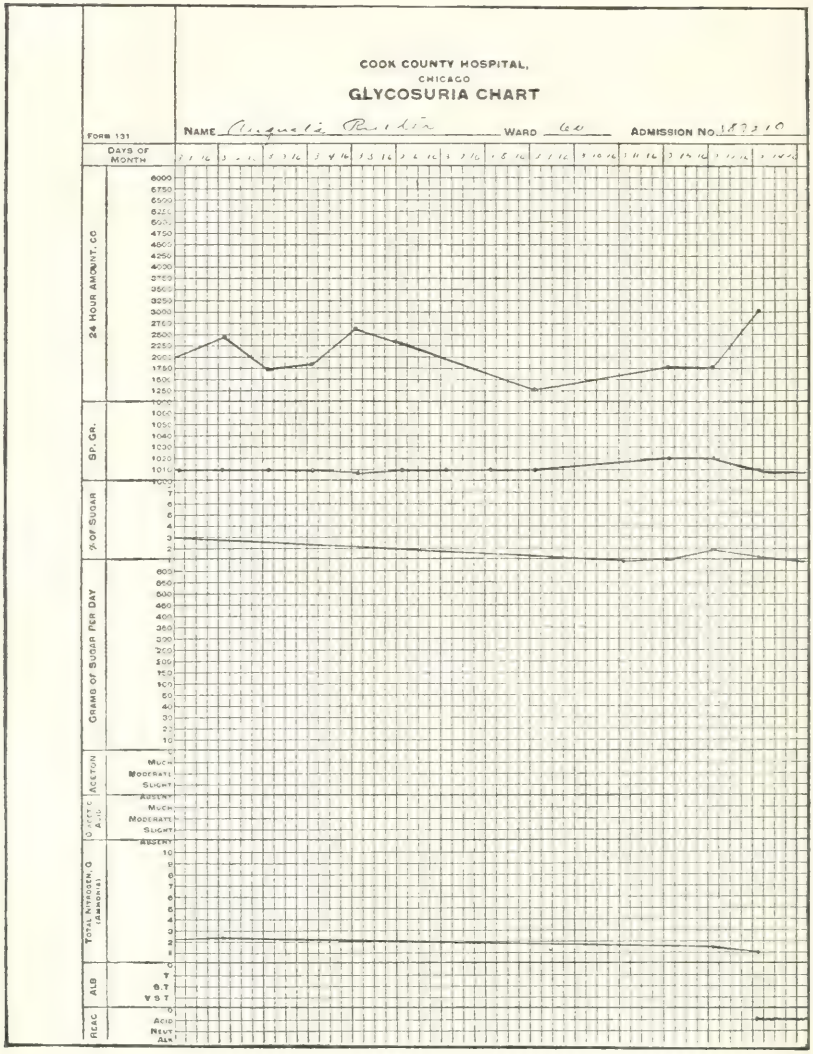

Fig. 3.

Because of these scars, especially the pigmented scar on the right leg, and also because of the history that she has given birth

to six children with only one living and has had one miscarriage, a blood-serum Wassermann test was made, but was reported negative. This is of some importance because of the possibility of a luetic infection causing a disturbance of the carbohydrate metabolism. Whether that is a disturbance of the sugar center in the nervous system or whether it is a luetic infection in the pancreas is a question. For that reason the Wassermann test was made, but was negative on the blood.

The blood-pressure has averaged 190 systolic and 90 diastolic.

Diagnosis.—From the symptoms and physical signs, assisted by the laboratory findings, the diagnosis is: first, diabetes mellitus; second, chronic interstitial nephritis; third, general arteriosclerosis producing, or at least associated with, myocarditis and emphysema; fourth, chronic peripheral neuritis; fifth, circulatory disturbance in the toes, particularly the middle toe of the left foot, with a distinct tendency to gangrene.

Case II.—Diabetes Mellitus with Gangrene of the Toes.

History.—*Present History.*—This patient is sixty-eight years old, and entered the hospital on the 14th of last November, when this history was obtained. About five years ago the patient began to lose weight and was told that she had diabetes. An intense itching appeared at that time in the region of the vulva and caused great discomfort. The patient has been doing housework up to the last week. During the last month the right foot has been very cold. Blisters have formed on both feet; one week ago an ulcer developed on the right leg. The patient previously had never been subject to boils, never suffered from pains in the extremities, and never had any excessive thirst or hunger. The bowels have been constipated for a long time. She urinates three or four times a day, and during the past week has been compelled to get up at night. There has been no cough, shortness of breath, chills, or fever.

Past History.—She had measles and whooping-cough during childhood, but no sore throat or rheumatism.

Family History.—Her father died of old age; her mother died in confinement. Seven brothers and one sister have all died. So far as can be determined no one died of tuberculosis, carcinoma, or diabetes.

Marital History.—She was married at twenty; her husband died seven years later of "inflammation of the bowels." She has had four children; one died of scarlet fever, one of dropsy, one cause unknown, one is living and well.

Habits.—She has used alcohol in moderation and denies venereal infection.

Physical Examination.—As in the previous patient, there is a rather loose skin and musculature, evidently some emaciation, with the rounded barrel-shaped chest due to emphysema. Examination of the heart reveals no apparent increase in the cardiac dulness and the sounds are clear, but there is an arrhythmia. The blood-vessels are hardened and stiffened. Abdominal examination is practically negative. The liver is palpable just below the costal border, but aside from that the findings are negative.

The Urinalysis.—The day she entered the hospital examination of the urine revealed the presence of albumin, a specific gravity of 1028, and the presence of sugar. Quantitatively determined, the amount of sugar at that time was 1.8 per cent. Frequent urinalyses, practically daily determinations since that time, reveal the continued presence of sugar. On the 25th of November the sugar reached 4 per cent. and that was on a rigid diet. On the 1st of December she was put on a starvation diet. The sugar continued in her urine for eight days following this period of starvation. Since that time she has been sugar free. A daily urinalysis has not revealed a trace of sugar, even with a gradual increase in the carbohydrates.

A Wassermann on the blood-serum was done on the 18th of November and reported negative.

Diagnosis.—First, diabetes mellitus; second, a local gangrene of the toes of the left foot and a localized gangrene of the posterior surface of the right leg, which healed spontaneously; third, emphysema and general arteriosclerosis.

Case III.—Diabetes Mellitus with Pulmonary Tuberculosis.

History.—*Present History.*—This patient is seventy-four years old and entered the hospital on the 16th of February. The following history was obtained through an interpreter: The condition started about four months ago with a slight cough, which was present principally in the morning, and with expectoration of a considerable amount of sputum. This cough continued for about a month, and then the patient noticed that she was drinking more water and passing more urine than normal. This was soon associated with an increase in appetite, especially for bread and potatoes, but in spite of that she began to lose weight, and this condition has continued up to the present time. The cough has gradually become more severe. She has never been troubled with itching or with pains or numbness or tingling in the extremities. She is occasionally annoyed with shortness of breath and palpitation, but has never had any swelling of the limbs.

Past History.—She had the usual diseases of childhood; she had some bladder disturbance, possibly a cystitis, early in life; she also met with an accident and suffered from fractured ribs three months ago. That was one month after the appearance of the present illness.

Family History.—The mother and father are both dead, cause of death unknown. She has no brothers or sisters.

Marital History.—Her husband and six children are living and well. She also gave birth to four other children who died from unknown causes.

Habits.—She has used alcohol only in moderation and denies venereal infection.

The Urinalysis.—The day she entered the hospital the urine showed a specific gravity of 1036, no albumin, but a positive reaction to Haines' solution. A Purdy test gave 3.3 per cent. sugar. Daily examinations of the urine up to the 24th inclusive showed constantly the presence of sugar, but gradually decreasing, so that on the 25th and 26th she was sugar free. On the 27th

there was a trace, but since that time she has remained sugar free.

Examination of the Blood.—The quantitative determination of the sugar content in the blood reveals by the Epstein method a percentage of .09. On the 19th of February a blood-serum Wassermann was done and reported negative.

The Temperature Chart.—The day she entered the hospital the first determination at 4 in the afternoon was 98° F. At 6 o'clock in the evening it was 100° F. The following morning the temperature was normal and remained normal all day. Subsequently there was a slight afternoon temperature of 99.6°, 99.4°, 99.2° F. by mouth. This, with the history of the cough, aroused our suspicion of a possible pulmonary infection.

Physical Examination.—Upon inspection, as you will observe, there is a very evident difference in the two sides of the chest. The right side is relatively retracted, and this becomes more evident with the forced efforts at breathing. The expansion is relatively poor, but is better on the left side. On percussion, there is good resonance on the left side above and below the clavicle. On the right side there is dulness, particularly above the clavicle. This dulness extends down to the lower border of the second rib anteriorly. We are unable to determine the respiratory excursion because of the lack of coöperation on the part of the patient. On palpation the tactile fremitus is increased over the area of dulness, and on auscultation the breath sounds are roughened, with mucous clicks.

It was on the physical findings that a diagnosis of pulmonary tuberculosis was made on this patient the day she entered the hospital. A standing order was left for a daily sputum examination, but no sputum could be obtained until the 26th of last month, ten days after her entrance, when a very small specimen was obtained. The examination of the sputum revealed the presence of numerous tubercle bacilli, establishing beyond any doubt the existence of a pulmonary tuberculosis of the right upper lobe.

Examination of the heart shows that the cardiac dulness is not increased. A very faint murmur is heard over the base of the heart, but no murmur is heard at the apex. The radial ar-

teries are not especially stiffened or hardened and the temporals are quite soft.

Examination of the abdomen reveals a relaxed condition of the skin and musculature. The liver is palpable two fingers below the costal border in the midclavicular line; the spleen is not palpable. The right kidney is palpable, the left is not. A mass is palpable in the umbilical region which is freely movable, slipping under the fingers, rather elongated, and giving the impression of a distended colon.

The **diagnosis** in this patient from the history and laboratory findings is: first, diabetes mellitus; second, pulmonary tuberculosis involving the right upper lobe; and third, a moderate degree of emphysema.

CASE IV.—DIABETES MELLITUS ASSOCIATED WITH PERIPHERAL NEURITIS, GENERAL ARTERIOSCLEROSIS, MYOCARDITIS, AND A GENERAL SPLANCHNOPTOSIS.

History.—*Present History.*—This patient, seventy-five years old, entered the hospital on the 5th of last November. She has no definite complaint, but was sent in by a physician for clinical observation and diagnosis. Her memory and mental condition in general are very much impaired and her statements are rather contradictory. She has had attacks of diarrhea alternating with constipation. She has also had some pains in the legs. She coughs occasionally, mostly in the morning, but has not expectorated any blood. She is not short of breath, but the feet have been swollen at times, particularly in the morning. Her appetite is good, but not excessive. She believes she has lost some weight. There is no history of headache or eye disturbance. She urinates frequently, sometimes every hour during the day and at least three or four times during the night. She thinks she is passing an increased amount of urine.

Past History.—She had measles and scarlet fever in childhood, inflammatory rheumatism early in life. Her history is otherwise negative.

Family History.—Her father and mother died of some type of paralysis. She had five sisters and four brothers, three of

whom are living and well. The cause of death of brothers and sisters is unknown.

Marital History.—The patient was married at twenty-eight. The husband died of paralysis which developed suddenly. She had eight children; three are living and well, four died in infancy, one at a later period from tuberculosis. There is also a history of two miscarriages.

Habits.—She has used alcohol moderately in the form of beer. She denies a venereal infection.

The Urinalysis.—The first urinalysis revealed a specific gravity of 1030, the presence of albumin and sugar. The percentage of sugar was not determined at the first examination. The following day a twenty-four-hour specimen, 2000 c.c., showed a specific gravity of 1026 with 1.8 per cent. sugar by the Purdy method. The urine contained sugar for a period of one week when, on a starvation diet, she became sugar free. She remained sugar free until December 7th. Then for three days there was a sugar reaction quantitatively of 0.4 to 1.6 per cent. She again became sugar free and remained so until December 24th, when she had 1 per cent. of sugar following immediately upon an error in diet. She was accidentally given a forbidden diet and promptly developed a glycosuria. That continued on the 26th, 27th, 28th, and 29th, the sugar going up to 4 per cent., but on the 31st it fell to $\frac{1}{2}$ of 1 per cent., and on January 2d it had practically disappeared. On the 3d no reaction was obtained. Since then she has remained sugar free except on two or three occasions, when there has been less than $\frac{1}{2}$ of 1 per cent.

A Wassermann test was made on the blood-serum of this patient and reported negative.

Diagnosis.—Physical examination, in conjunction with the symptoms and laboratory findings, justifies the diagnosis of diabetes mellitus associated with a mild type of peripheral neuritis, general arteriosclerosis producing or accompanying a myocarditis with a mitral incompetency, emphysema, and general splanchnoptosis. The liver is low, the spleen and kidneys are palpable, and there is a dilated condition of the stomach.

Case V.—Diabetes Mellitus with Gangrene, Necessitating the Amputation of Two Toes.

History.—*Present History.*—This patient, forty-nine years of age, entered the surgical service of the hospital on October 23, 1915. The history states that she was admitted because of a discharging sinus of the left foot and an abscess in the left temporal region. The patient left the hospital last August after the amputation of two toes. The wounds were nearly healed at that time. Two weeks ago the left foot became painful, finally suppurated, and has been discharging pus and blood since. The wound at times has a disagreeable odor. The foot and leg have become swollen and painful, particularly when she is up and about. About one week ago she noticed a pimple over her left ear. This increased in size and finally ruptured spontaneously and discharged pus. She noticed no fever and had no chills. She has had pains in the back, excessive thirst, and has passed more urine than normal.

She was treated on the surgical service from October 23d until the 27th, when she was transferred to the medical service on the diagnosis of diabetes and ulcers.

The *history* when she entered the medical service was as follows: Two and a half years ago the patient was in this hospital with painful discoloration of her toes, and was told that she had diabetes. She has not been well since that time. In August, 1915, the toes again became painful and discolored and were amputated. She was put upon a strict diet and left the hospital feeling comparatively well. Since leaving the hospital she has been on a more liberal diet. Her foot again became painful, commenced to discharge, and three days before entering the hospital she developed an abscess in the temporal region.

Past History.—She had pneumonia fourteen years ago, typhoid fever twelve years ago, rheumatism seven years ago. Personal and family history are negative.

Physical Examination.—The lungs reveal nothing abnormal. There is a slight increase in the cardiac dulness to the left. The blood-pressure registers 120 systolic, 80 diastolic.

Examination of the feet at that time revealed practically the same condition which exists today, *i. e.*, absence of the second toes and a slight discharge, which has now practically disappeared.

The Urinalysis.—The urine has revealed the presence of a trace of albumin from time to time without casts, and the presence of sugar, which has varied quantitatively up to November 2d. On that day she became sugar free. On December 12th there was just a trace, and on the 24th, 25th, and 26th again a trace of sugar on a gradually increasing amount of carbohydrates. At present she is sugar free. She has been passing quantitatively about 3000 c.c. of urine, but with no sugar and a specific gravity of 1018 to 1020.

This constitutes, then, a group of five cases of diabetes mellitus with complications.

The first patient presented had a diabetes mellitus with a chronic interstitial nephritis, arteriosclerosis, chronic peripheral neuritis, and beginning gangrene of the toes; the second one had a diabetes and gangrene of the toes of the left foot; the third one a diabetes with pulmonary tuberculosis; the fourth one a diabetes with a peripheral neuritis, general arteriosclerosis, myocarditis, and splanchnoptosis; and the fifth one a diabetes and gangrene of the toes. Of all the complications which occur in diabetes mellitus, gangrene and pulmonary tuberculosis are the most frequent. We have to keep in mind, of course, the relatively high percentage of diabetics who die with a coma, but this is rather an accident than a complication. Of the frequency of this occurrence we will give some figures later. In cases where pulmonary tuberculosis is a complication they usually run a rather rapidly fatal course.

Additional Notes on Cases of Diabetes

April 17th.

You will recall that we presented a few cases of diabetes a couple of weeks ago. One of these presented also the clinical evidences of a nephritis. She had the urinary findings and blood-pressure indicative of a kidney involvement. The systolic press-

ure was over 200, and at the time she was presented she also showed sugar in the urine. We had the greatest difficulty in obtaining a sugar-free urine in her case. Since that time her condition has not progressed favorably, and yesterday morning on making the rounds it was found that she had a complete left-sided hemiplegia. The patient was not aware of this condition until she awoke and found that she was unable to move her left arm and leg. The question of the etiologic factor, whether it was a hemiplegia due to an embolism or to a thrombosis or to a cerebral hemorrhage, we have not yet determined.

The patient with the pulmonary tuberculosis was transferred to the tuberculosis department. The two patients with gangrene were under observation for several days after being presented, still remaining sugar free, and both have been transferred to other departments for the treatment of the complications.

DIABETES MELLITUS WITH ACIDOSIS

Clinical Picture of Diabetic Coma. Causes of Death in Diabetes. Fats the Dangerous Elements in the Diabetic Diet. Prevention of Acidosis Essential in the Treatment of Diabetic Coma. Proper Method of Applying Starvation Treatment. Futility of Medicinal Treatment After Coma has Developed.

March 13, 1916.

History.—*Present History.*—This patient is forty-four years old and entered the hospital on March 11th. She gives the following history: Four days ago, following a cold, she was seized with a very severe headache and earache accompanied with a fever. The pain has been so severe that the patient could not sleep. The day she entered the hospital the ear began to discharge and the patient felt better. Nine years ago she began to notice an increase in hunger and thirst, accompanied by an increase in the amount of urine voided. She was told she had diabetes and was placed on a diet. She has decreased in weight from 194 to 114 pounds at present and her strength has gradually failed. She has had severe headache, her vision has failed somewhat, and she often sees flashes of light before her eyes. During these nine years she frequently had fainting spells which have recently increased in frequency. She can usually tell when an attack is coming on, though she sometimes has fainted and received bodily injury. Convulsions have occurred from time to time, each attack usually lasting half an hour. During the last two years she has had attacks of generalized furunculosis. Itching of the skin has been present much of the time. Last year she had a severe attack of pruritus vulvæ.

Past History.—She was always well and strong until nine years ago. She had measles in childhood, but no other illness. The family history is negative.

Marital History.—She married at the age of thirty-two and has four children, all living and well. There is a history also of

two miscarriages at approximately the sixth month and several which occurred earlier. She has been pregnant thirteen times in all. There is no history of the use of alcohol.

Temperature Chart.—When she entered the hospital the temperature was 99.4° F., the pulse 88, respirations 28. On the following day the temperature in the morning was 99.4° F., in the afternoon 99.6° F. The next day there was a normal temperature. This morning, by mouth, it was 97.8° F. She has had no pyrexia of any consequence since she entered the hospital.

Physical Examination.—One of the first things that attracts the attention when one approaches the patient is the sweet odor of her breath, a condition of some significance. This is particularly noticeable when the patient is in one of the private rooms. Even here in the amphitheater the odor is easily detected. Another condition which you must have observed is a tendency to stupor. The patient is drowsy, and that is not due to medication. She has received no opiates of any kind since she has been under observation. You will recall, too, that previous to her admission to the hospital she was suffering from headache and a discharging right ear, evidences of which you can notice on the pillow and also in the external auditory canal. It is a rather profuse discharge in spite of repeated irrigations. On the opposite side the same condition is also present. So we should bear in mind the possible relation of this stupor to the ear condition or some complication, particularly a meningitis.

On further general inspection of the external cutaneous surface we find evidences of traumatism, probably due in part to scratching, also remembering that the patient has suffered from repeated attacks of generalized furunculosis. Perhaps some of these pigmented areas are due to the healed local infections. We will first direct our attention to the cardiac and pulmonary condition and then return to the nervous system.

Cardiac Findings.—On inspection, palpation, and percussion there are no abnormal findings. There is a very distinct systolic murmur at the apex, a definite systolic murmur over the aortic area. The murmurs are quite circumscribed and not transmitted.

Pulmonary Findings.—On inspection and percussion nothing

abnormal is revealed. On auscultation a few scattered râles can be heard over either side. There is no change in the respiratory murmur. Posteriorly the skin shows evidence of local infections and a very slight edema is present. It is impossible to put her in the upright position on account of her general condition. A few râles can be heard posteriorly.

Abdominal Findings.—The abdomen is very lax; there is no tenderness or discomfort on palpation. The liver is not palpable; the spleen is not enlarged on percussion and is not palpable. The lower pole of the right kidney is palpable, the left is not.

Extremities.—Examination of the lower extremities shows that there is a slight edema over the anterior surface of the tibia. No patellar reflex can be elicited, at least not without reinforcement. There is no Babinski, nor is a Kernig present on either side.

Head and Neck.—There is no rigidity of the cervical muscles and little discomfort on extension or flexion. There is no resistance to any movement of the head. There is no external ocular paralysis, no strabismus, no nystagmus; the pupils are equal and respond to light rather promptly. There is tenderness over the right mastoid, but no swelling or edema. There is a greater amount of tenderness and a slight amount of edema over the left mastoid. Only moderate pressure in this region causes discomfort. There is no cervical adenopathy; the lips and tongue are dry. The patient has a complete upper plate, all of the teeth having been removed. The lower gums and teeth present a condition of pyorrhea. The gums are red. There is purulent material about the roots of the teeth which are all extremely loose. The whole pharynx is dry and red with an accumulation of débris.

Laboratory Findings.—A single urinalysis made the day she entered the hospital, a catheterized specimen, showed a specific gravity of 1022, straw color, acid reaction, albumin double plus, sugar one plus; diacetic acid was present in abundance. Pus cells and granular casts were found microscopically. A more complete urinalysis has been attempted, but it has been difficult to obtain a twenty-four-hour specimen because the patient is involuntarily voiding her urine. The examination of the specimen

this morning also reveals a large amount of diacetic acid; the ammonia content was not estimated because the twenty-four-hour specimen was not complete.

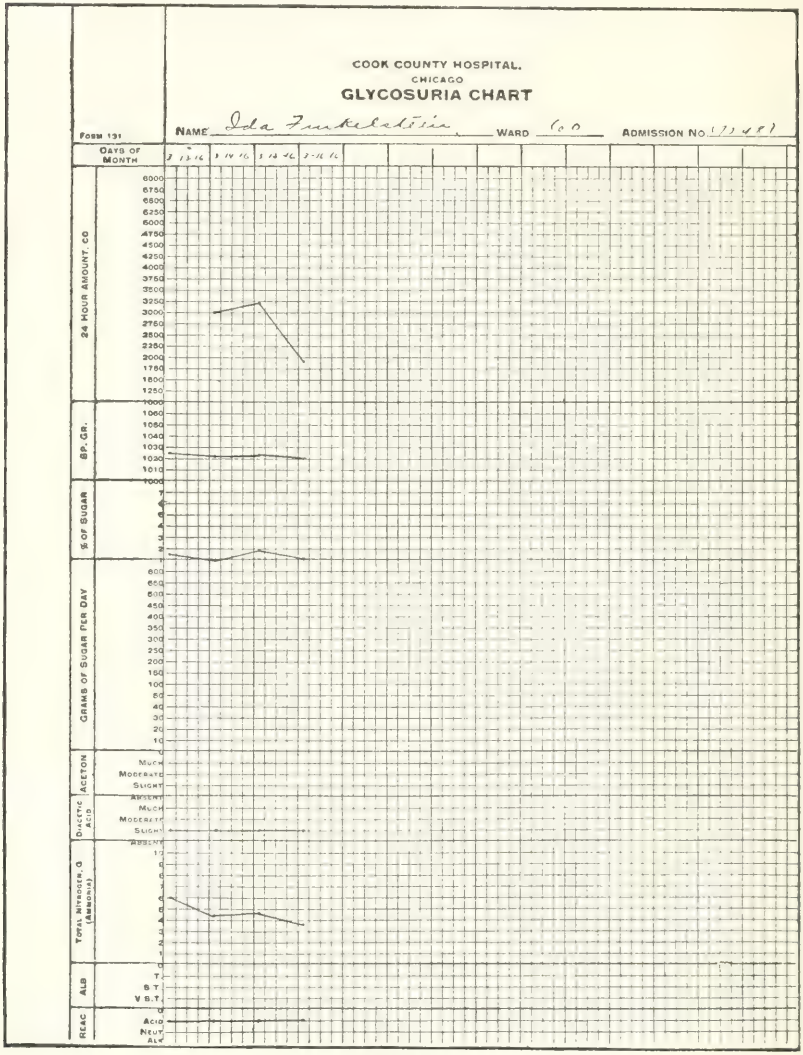

Fig. 4.

Blood Examination.—The white count is 9200; only a slight increase. Blood was taken for a Wassermann and reported negative.

General Considerations.—The patient was presented in view of the 5 cases of diabetes shown a week ago, and particularly because of the possibility of a diabetic coma. It is true that her history must be considered. She tells us that she has previously had repeated attacks which were interpreted as convulsions. Did she have convulsions, and, if so, what type of convulsions were they? Were they hysteric attacks, or due to a nephritis, or possibly due to her diabetes—for the history tells us she has had a glycosuria for nine years—or were they due to some other cause? We would conclude that these convulsions were probably not hysteric, judging at least from the personal history of the patient and the urinary findings. In spite of the history of glycosuria and the presence of a large amount of diacetic acid, it seems impossible to account for her convulsions on the basis of the diabetes, owing to the fact that if a coma develops it is not recurrent. On the other hand, the presence of an abundance of albumin and of granular casts justifies us in assuming that these convulsions were uremic in character. Their exact nature, of course, is difficult to determine.

At present we have to consider chiefly the possibility of the stuporous condition being the result of the otitis media, a complicating meningitis, or an impending diabetic coma or acidosis, that is, a retention of various acids due to a disturbed metabolism. Proof of the latter is the presence of sugar and diacetic acid, the strong, sweetish, sickening odor of the breath, and the definite drowsiness.

In this connection it will be of interest to refer to a recent article by Joslin, of Boston. From 1894 up to the 15th of last December Joslin has observed 945 cases of diabetes; of these 425 died. It is instructive to consider the mode of termination. How did these patients die? You will remember that of the 5 cases of diabetes presented a week ago today, one had a cardiovascular involvement, one of them an interstitial nephritis, two had gangrene, and one pulmonary tuberculosis. Since that time examination of the sputum has revealed a marked increase in the number of bacilli. On account of the free elimination of bacilli and the possibility of infection of the other patients in the

ward it was considered advisable to transfer her to the tuberculosis department. We remarked at that time that tuberculosis was not an infrequent complication of diabetes and that it usually ran a rapidly fatal course. We have at present in private practice two other cases of diabetes with pulmonary tuberculosis, one of which is running a rapid course.

Of Joslin's 425 cases which terminated fatally, 273 died in diabetic coma; that is, a little over 64 per cent. Of the remaining number, 52 died with cardio-renal-vascular involvement; that is, arteriosclerosis, cerebral hemorrhage, decompensated heart or nephritis. Of the remainder, 36 died from some acute infection; 16 from pulmonary tuberculosis; 17 died of carcinoma—uterine, gastric, cutaneous or some other type—and 16 died of various causes.

So in the consideration of the prognosis and treatment we have to keep in mind the possibility of a diabetic coma. In this patient there is a bilaterial otitis media, but no evidences of meningeal or cerebral involvement. To diagnose a meningitis or cerebral abscess to account for this definite stupor is impossible. Since her stay in the hospital her temperature has been practically normal and the leukocyte count has not materially increased. On the other hand, you must remember that in cases of diabetes in which a local infection occurs there is constantly a lowering of the carbohydrate tolerance. After every infection, furunculosis, etc., clinical experience teaches that the carbohydrate tolerance is lowered. With this fact in mind, we might believe that through local infection, which may have started as a throat infection with extension to both ears, her carbohydrate tolerance was lowered with disturbed metabolism and there resulted an impending coma.

It was with this idea in mind that the patient was brought into the clinic, and more particularly to emphasize that the prevention of an acidosis is the essential and important treatment of a diabetic coma, for the reason that after a diabetic coma has developed it terminates fatally. In not a single one of the numerous cases of diabetic coma that we have observed has there ever been more than a temporary improvement for a day or two and then a fatal termination.

TREATMENT OF DIABETES WITH SPECIAL REFERENCE TO ACIDOSIS

It is believed at present that acidosis results from the retention of various acids. The carbohydrates play only an inferior rôle if any in the production of this condition. The belief is entertained that acidosis results largely from an increased intake of fats. This is an important element in the treatment of diabetes, and particularly with reference to the starvation treatment. Diabetics with an acidosis, if placed at once on a complete starvation treatment, are in great danger of developing a coma. How are we, then, to avoid the precipitation of a coma? The better plan to pursue is, first, if conditions will permit, to keep the patient on the accustomed diet in order to determine the urinary findings. Then, instead of at once placing the patient upon absolute starvation, decrease the amount of fats. After a day or two eliminate all of the fats, the patient still taking the same amount of carbohydrates and proteins as when on the full or usual diet. In another day or two the proteins are eliminated because it is believed that an acidosis may in part result from the protein intake. After the fats and proteins are eliminated reduce the carbohydrates daily by one-half until 10 grams only are given. Then the patient is ready for an absolute starvation diet. In this manner even a threatening acidosis may be prevented by gradually eliminating the fats and proteins and then the carbohydrates, keeping the patient on a starvation diet until the urine is sugar free, and then gradually increasing the carbohydrates, proteins, and fats until his tolerance has been determined.

It is a clinical experience verified by many that acidosis may be precipitated simply by increasing the fats, the butter or meat fats or the amount of olive oil. It is quite generally believed that the fats are the dangerous elements in the diabetic diet; therefore, if by proper regulation of the diet the development of an acidosis can be prevented, far more will have been accomplished than can be hoped for after a coma has developed.

After the acidosis is well developed then the chief treatment, aside from the question of diet, is the *medicinal treatment*. It is well known that when coma has developed we no longer restrict

the diet, but place the patient at once on a full diet, even giving glucose by mouth, per rectum, or intravenously.

Joslin believes that if the sodium chlorid is decreased there is greater danger of the development of coma. He also believes that the liberal administration of sodium bicarbonate is followed by rather indefinite results, and he questions the advisability of giving sodium bicarbonate, but rather favors allowing the patient to neutralize the acidosis by his own ammonia.

Further Notes, March 20th.—You will recall that one week ago today we presented a case of diabetes with an otitis media. At that time we considered some of the probable causes of her drowsy condition and concluded that it was a precomatous state of a diabetic coma. After the patient left the clinic she was examined by an ear specialist who found no definite mastoid involvement and advised against operation. She was placed on treatment, with possibly a slight improvement on Tuesday and Wednesday. On Thursday, Friday, and Saturday her condition became worse. Yesterday (Sunday) morning there was a very definite increase in the intensity of her stupor. It was impossible to rouse her and she complained of headache and backache. During the morning nausea and vomiting developed and she no longer retained any food. As previously related, this is one of the untoward symptoms in a diabetic. As soon as the nausea and vomiting occurred it was apparent that the termination was near. Preparations were at once made to give intravenously 300 or 400 c.c. of a 4 per cent. solution of sodium carbonate and also to give glucose per rectum. Before the preparations could be completed the patient suddenly died.

This was the outcome of a diabetes with coma. When presented in the clinic there was only the precomatous condition, which went on to a true coma in spite of treatment.

This is only another illustration of the usual termination when coma develops. In the fairly numerous cases admitted in coma, even by vigorous and early treatment not more than a temporary improvement has occurred. Medical treatment of a diabetic coma is futile.

CLINIC OF DR. SOLOMON STROUSE

MICHAEL REESE HOSPITAL

DIABETES AND SURGERY; DIABETES AND PREGNANCY. THE TREATMENT OF DIABETIC ACIDOSIS

Three Special Dangers in Operating Diabetics. Best Method of Preparation of Diabetic for Operation. Co-operation of Surgeon and Internist Desirable. Local Anesthesia Advisable Whenever Possible. Analysis of 338 Cases. Danger of Diabetes in Pregnancy.

April 19th.

CASE I.—This patient, thirty-eight years old, a Jewess, came to the hospital on the 28th day of December, 1915, as a patient of Dr. Irving Stein, complaining of "burning sensation in the vagina, blisters around the external genitalia, pain in the lower abdomen and back." These symptoms have been present about ten months and they have gradually grown more intense. A leukorrheal discharge is also present. On standing she notices that her abdomen becomes markedly distended. The menstrual history is negative. She has three children living and well. There is nothing of importance in the family history.

The *general physical examination* shows a well-built, somewhat flabby woman. The lungs and heart are negative. The abdomen shows a mass deep in the pelvis easily palpable through the soft abdominal wall. This, on vaginal examination, is shown to be the uterus with multiple fibroids.

The patient came into the hospital for operation, and sugar was discovered in the urine when a routine preoperative urinalysis was made. Then, upon requestioning, the patient admitted (although she *did not complain of*) a *marked* increase in thirst for about three years, accompanied with increased urination and

burning sensation at the vulva. No other symptoms could be elicited. The diagnosis of diabetes mellitus was made by the establishment of the sugar as dextrose by means of the polarization and fermentation tests. As soon as the diagnosis of diabetes was established operation was postponed. The patient was then put on a strict carbohydrate-free diet plus 150 grams of bread. The *urine* on this diet showed the following (Fig. 5):

Amount in twenty-four hours, 1200 c.c., specific gravity 1028, acid reaction, no albumin, 2.5 per cent. or 30.5 grams (1 ounce) of sugar, trace of acetone and no diacetic acid; ammonia excretion was .65 gram.

Two days of complete starvation rendered the urine both sugar free and acetone free. The diet was then changed by adding vegetables containing 5 per cent. carbohydrates, eggs, with the gradual addition of vegetables richer in carbohydrate, and meat, until finally at the end of about ten days she was getting a diet containing 85 grams of carbohydrate in the following form: Bread, 75 grams; apple, 1; 250 grams of 5 per cent. vegetables; 100 grams of 10 per cent. vegetables.[1] Bicarbonate of soda was administered for two days before the operation both by mouth and as rectal infusions. The urine for five days before operation showed absolutely no acetone, and for fifteen days before operation had shown no sugar. The specific gravity was 1012, ammonia excretion .52.

On the 17th of January Dr. Lester E. Frankenthal performed a vaginal hysterectomy, right salpingectomy, and oöphorectomy; the anesthetic was gas preceded by hypodermic injection of morphin and atropin. The uterus was four times its normal size and was filled with fibroids. The patient returned from the operating room in excellent condition, and was placed on the usual postoperative treatment, namely, nothing but water the first day, with a slight amount of tea and broth the second day; the third day rice and milk were added. Then the diet was gradually increased to practically what she had been receiving the day before

[1] For convenience in expression vegetables and other articles of food are designated as "5 per cent.," "10 per cent.," etc., depending on the amount of carbohydrate content.

Admission No. 84100

Name MRS R. C Ward 457 Room

MICHAEL REESE HOSPITAL

SPECIAL DIABETIC CHART

	DEC. 29	30	31	JAN 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
MEAT	+	+							50	50	50	50	50	50													50	50
EGGS	3	3	2		2	2	3	3	3	5	5	5	5	5									1	2	2	2	2	2
VEGETABLES 5%	+	+	+		150	300	300	300	300	250	250	250	250	250		DIET							50		100	100	100	100
" 10%	+	+					50	50	50	100	100	100	100	100		NOW NOT												
BREAD	150	150	50							25	40	55	75	75		CHANGED								60	60	75	75	75
BUTTER	+	+						20	20	40	40	40	40	40												20	20	20
CREAM	+	+						20	20	30	30	30	30	30											80	80	80	80
CHEESE	+	+					20	20	20	50	50	50	50	50										50	50	50	50	50
BACON	+	+					20	30	30	30	30	30	30	30														
NUTS								20	20	20	20	20	20	20		URINARY												10
OLIVES										10	10	10	10	10		FINDINGS												
CEREAL																CONSTANT												
BRANDY			30	50	50	50																100	150	200	200	200	200	200
PROTEIN								37.5						85														54
FAT								62						120														49
CARBOHYDR								16.						81														102
CALORIES								820						1810														1080
URINE																												
SUGAR-Gm	28	30	10	0	0	0	0	0	0	0	0	0	0	0						++	0	0	0	0	0	0	0	0
ACETONE	+	+	+	0	+	+	+	+	+	0	+	+	+	0						0	+	++	+	+	0	0	0	0
AMMONIA	.65	.65	.2	.27	.32	.58	.62	.64	.43	.24	.43	.52	.44	.24						?	6	.34	.42	.32	.46	.46	.42	.43

OPERATION

Fig. 5.—Plus (+) mark signifies that the particular food was given, but was not measured. Except when following "eggs" the numbers refer to grams or cubic centimeters (30 grams figured as 1 ounce; 30 cubic centimeters as 1 fluidounce). The figures in the "eggs" column refer to *number* of eggs, not to weight. The food values in protein, fat, carbohydrate, and calories are approximate computations from published analyses. In giving the specific gravity of the urine the two last figures only are written. The amounts in the ammonia column are actual grams of ammonia per day, as measured by the simple formalin titration.

operation. The urine had shown a large amount of sugar for *one* day after the operation. This entirely disappeared and remained absent until the patient was discharged from the hospital. An acetone reaction was present in a slight degree for four days after operation. The patient at no time exhibited any manifestations of diabetic acidosis or of postoperative shock. She left the hospital thirteen days after operation in excellent condition, with no sugar or acetone in the urine and with a completely healed wound. Two blood-sugar examinations had been made during the patient's stay in the hospital, one before the operation and one after, and in both instances showed 0.16 per cent., a slight but definite increase over normal.

This case brings up for discussion many points of interest in the complex problem of the interrelation of diabetes and surgery. This patient's symptoms were entirely referable to her uterine fibroids. Incidentally, it might be remarked that many individuals have diabetes for years without *noticing* the increase in thirst and urination until some complication or some other disease causes them to seek medical aid. The intensity of the secondary symptoms makes them forget the mild primary disturbance of increased thirst and increased urination, which frequently are omitted from the history entirely, and are elicited only after the diagnosis of diabetes has been made. The importance of this fact, especially in surgical cases, cannot be overemphasized, and is a definite indication for careful urinalysis in all surgical cases. The frequent discovery of unsuspected diabetes at life insurance examinations substantiates this argument.

This patient had fibroids for two or three years, but when the operation was about to be performed she had both sugar and acetone in the urine. There was no haste for operating, and she affords an excellent example of that group of diabetics in whom operation ought to be performed not as an emergency measure, but to prolong life or to increase usefulness. We shall see from our subsequent classification that such a patient offers the very best hope for surgical interference, but such hope can be realized only when there is complete co-operation between internist and surgeon. The careful preparation which this patient received

practically converted her into a normal surgical risk. However, had she been treated in a routine way by a so-called "diabetic diet" we might have increased the possible dangers from the operation many times. We shall come back to this in a moment.

Surgery of the Diabetic.—This can be considered from two different points of view. One can either classify the cases from the standpoint of surgical urgency, namely, into (1) emergency cases and (2) those cases in whom the operation is not urgent. Or one can divide the patients into two groups on a different basis: (1) those cases in whom the surgical indication is caused by the diabetes; and (2) those in whom the surgical indication has nothing to do etiologically with the diabetes. It is well to subdivide the last group of cases further into (*a*) those in whom the surgical operation is associated with an infection and (*b*) those in which there is no infection. Acute suppurative appendicitis or infected gall-bladder in a diabetic would belong to the first subdivision, while carcinoma of the breast or fibroid of the uterus would belong to the second. Obviously, either class of this latter division could group emergency cases, so that the two classifications actually dovetail.

Naturally, the surgical procedure in a case of diabetes would depend entirely into which group the case fell. Surgical emergencies necessary to save life would mean immediate operation despite the diabetes. In such cases delay for medical treatment is even more dangerous than it would be for a non-diabetic, as, for example, in an appendiceal abscess. But when the surgical indication is not so urgent, both physician and surgeon must bear in mind that the diabetic always is a difficult subject for operation. Why? Because he is threatened with *three special dangers*. The first of these is the danger of wound infection. Modern aseptic surgery has proved, however, that the danger of infecting a diabetic is not great provided *rigid* asepsis is used. But if even a few micro-organisms are introduced into the wound there is a tendency toward suppuration much more marked than in the normal individual. The cause of this has been investigated by numerous workers. Very probably the tissues

of a diabetic have been damaged by the impoverished blood and, therefore, rendered less resistant to infection. Hardmann has investigated blood from the various aspects of immunity, and has come definitely to the conclusion that there is no change in the immune bodies of the diabetic which can be attributed in any way to the increased blood-sugar always present.

The second danger of the diabetic, though rather negligible in the presence of aseptic surgery, is the danger of non-healing of wounds. Occasionally in a diabetic in whom an urgent operation is performed non-healing of wounds and hemorrhage may occur. This probably is due to the same factor that has just been discussed under the question of infection. But neither one of these two elements can be compared in the severity of their potential danger with the danger of *acidosis* and *coma*. A diabetic, no matter how well treated, no matter how skilful the surgeon, no matter whether the anesthesia be local or general, may, despite everything, develop a postoperative coma. The danger is always present, and in operating on a diabetic every possible means should be used to guard the patient against this danger.

Preparation of a Diabetic for Operation.—Now, as illustrated in our patient, the preparation of a patient for operation is possible. The principle underlying any procedure is the same, whether the diabetes is of the mild or the more severe type of the disease, and that principle can be stated as follows: The danger from coma in a diabetic is definitely due to one ultimate thing, and that is the inability of the diabetic organism to burn carbohydrates. No matter what the immediate cause of acidosis may be—and there are many theories of acidosis—the final factor is the carbohydrate factor. Therefore, without doubt, the correct principle in all cases is to aim at *making the patient utilize carbohydrates if possible*. Now how can this be accomplished? The details of dietetic therapy in diabetes vary in every case, so that one can give only general rules which must be modified to suit the individual. However, it can be stated that the only way to make a patient burn carbohydrates is first to get his urine free from sugar.

The method of procedure that seems to me the most logical is, first of all, to place the patient on a standard diet containing a known amount of carbohydrate. Under this standard diet one can in two days ascertain the severity of the disease. The procedure then would be to use means to render the patient sugar free and acetone free. Allen's starvation method is advisable in the more severe type of cases. In the less severe type I think it is preferable to employ more conservative means, such as gradual reduction of carbohydrate until the urine is sugar free. In either case, after the urine has become sugar free, the next step is to run the carbohydrate content up to as high a point as possible without producing glycosuria. I know of no better way of doing this than by the gradual addition of carbohydrate, protein, and fat to the diet as recommended by Allen and as illustrated in our case. If a patient is burning enough sugar to have no acetone in the urine, and if the ammonia excretion does not indicate any disturbance of the alkali balance, a patient can be considered ready for operation. Most men believe in saturating the patient at this stage with alkali. The advantages of alkali therapy lie in the fact that the sodium salts of the beta-oxybutyric acid are more easily eliminated by the body than the acid itself, and, as far as we know today, beta-oxybutyric acid is the acid of importance in diabetic acidosis. Another factor of advantage in the giving of alkali is that any possible alkali starvation is taken care of. In this connection Van Slyke's new method of determining the carbon dioxid reserve of the blood-plasma will become of value as a pre-operative test of the dangers to be encountered.

The attainment of this ideal state is, of course, not always possible, and the indications for action may not be so clean-cut. But in the very severe cases the only operations allowable are those without which life is endangered. A diabetic gangrene spreading rapidly, a carbuncle with symptoms of sepsis, a ruptured appendix are surgical indications despite severe diabetes. In the less severe cases, when the urine cannot be made completely sugar free and acetone free, the principles of pre-operative and postoperative diabetic therapy can still be applied, provided

it is remembered that mere reduction in carbohydrate intake is not the aim of therapy. Such cases can usually be made sugar free, and then they will utilize enough carbohydrate to ward off a large part of the danger of coma, even though they pass a small amount of sugar in the urine. Naturally, in cases of any severity, judgment is required in balancing the dangers from the operation with the dangers that would arise without operation, in exactly the same way as judgment is required in operations on patients with heart disease.

Granted, then, that a patient has been prepared in the best possible manner, the next factor that must be considered is the *anesthesia*. Most surgeons believe that whenever possible local anesthesia is indicated, and chloroform is always contra-indicated. The tendency at present is to use nitrous oxid in all cases of diabetes where the condition of the cardiorenal system does not contra-indicate its use. Not sufficient data are available on this point to allow statistical conclusions, but from the theoretic standpoint an analogy of value may be drawn between the surgery of the diabetic and surgery in children. As is well known, children are prone to react to anesthesia by the production of more or less severe acidosis. The work of Dr. Ira Frank of this hospital has shown that the postoperative acidosis of children is less likely to follow nitrous oxid anesthesia than ether, and if it does occur it is likely to be less severe. Since, then, children react to anesthesia with acidosis and diabetics react in the same way, the analogy might support the current tendency to employ nitrous oxid whenever possible.

Now, as far as surgery itself is concerned, conservatism is the keynote of success. Not being a surgeon, I shall not discuss the technical problems, but an analysis of the experience at the Michael Reese Hospital, as shown in the following chart, may prove interesting. I have been allowed access to the records through the courtesy of many of my colleagues, whose kindnesses and my obligations are both apparent.

DIABETES

	Total number of cases.	Deaths, per cent.
	338	17.8
Carbuncles, gangrene (not operated)	13	30
Total operations	38	31.3
For carbuncle, gangrene, etc.	22	27
Peritonitis	2	100
Non-diabetic conditions	14	28
Total prepared operated cases	15	13
Prepared non-diabetic conditions	8	0

In the last fifteen years there have been 338 cases of diabetes at the Michael Reese Hospital, with 17.8 per cent. deaths; among these there have been 38 operations of all kinds, with total deaths of 31.3 per cent. If we classify these cases according to the scheme outlined above we find 22 of the operated cases were operated on for carbuncles, gangrene, and similar sequelæ of diabetes, with a death-rate of 27 per cent. The danger in extra-diabetic infections is illustrated by the two patients with appendiceal abscess and peritonitis, both of whom died. There were 14 operations for fibroids, enlarged prostate, carcinoma, etc., conditions having nothing whatever to do with the diabetes, with a death-rate of 28 per cent. It is extremely important, however, to note that *8 of these patients who had been properly prepared for operation survived.*

This experience is also well exemplified in the current literature. Karewski, of Berlin, a man whose surgical experience has been tremendous, reports 225 operations on diabetics with a total death-rate of only 21 per cent.; a death-rate from coma of 15 per cent. He had among these 225 cases 68 extradiabetic conditions that were not associated with infection, with a death-rate of only 11.8 per cent. Karewski worked on the principle of treatment outlined in this discussion and his results speak for themselves. Compare them with Fischer, also a Berlin surgeon. Fischer reports 86 operations on diabetics, with a death-rate of 48.8 per cent. Fischer does not believe in co-operation. He has a death-rate over 100 per cent. higher than Karewski. Furthermore, Philips has collected a large series of statistics, and has shown that prepared cases have a mortality of 17.7 per cent.,

while those that were not prepared show a death-rate of 36.4 per cent.

Now let me translate from a symposium on diabetes and surgery reported by Krause in 1914: "We must go closer into the manner of getting a patient with diabetes free from sugar, since this is not without influence on the appearance of coma or on operative statistics. It seems to me that the position of diabetes as a contra-indication to operation can be exactly established only after we learn which diabetics usually die and under what influences they die."

Payr has this to say:

(1) Diabetes is not always a contra-indication to operation.

(2) Those cases of diabetes which show both acetone and diacetic acid, or beginning coma, are the only absolute contra-indications.

(3) Abscess, carbuncles, gangrene in diabetes must be operated on even under the second condition.

(4) Aseptic wounds heal as well in diabetics as in non-diabetics.

(5) To determine the severity of the diabetes we must know:

(*a*) The amount of sugar present.

(*b*) The ease with which the sugar can be made to disappear.

(*c*) The ease with which the patient's tolerance for sugar is increased.

(6) *Whenever possible prepare a patient.*

(7) The operations which, despite the diabetes, eventually must be done are:

(*a*) Incarcerated hernia, appendicitis, etc.

(*b*) Indications which would later threaten the life of the patient, such as, for instance, cancer.

(*c*) Conditions which interfere with the usefulness of the patient.

(8) Cosmetic or plastic operations on diabetics are always contra-indicated.

(9) All operations must be done with (*a*) least possible anesthetic; (*b*) the operations should be made technically as simple

as possible; (*c*) exact asepsis; (*d*) operations on the limbs should be done under compression of big vessels with immediate stoppage of local hemorrhage.

(10) The prognosis must always be "reserved."

(11) The postoperative diet is important.

(12) No diabetic with dangerous surgical disease should be prevented from being operated upon because he is a diabetic.

One other point of importance is that when a diabetic is to be operated upon the less fuss made about it the better. It must be remembered that a diabetic shows a strange susceptibility to psychic impulses. Any emotion may produce glycosuria in a diabetic. It therefore seems clear that a diabetic who *must* undergo an operation should be encouraged and cheered in every possible way.

CASE II.—DIABETES AND PREGNANCY. THE TREATMENT OF ACIDOSIS

This patient is a married woman, twenty-eight years of age, who was referred to me through the courtesy of Dr. J. B. DeLee. In January, 1915, having some eye trouble, she consulted a doctor, who found on examination of the urine 5 per cent. sugar. Although she dieted very little the sugar did not persist constantly. In July, 1915, she became pregnant. After pregnancy started her capricious appetite was such that she did not diet in any way. In November she consulted Dr. DeLee for the pregnancy and he called me in to see the patient. She had one child nine years of age and had been in perfect health all the time. She was a very highly neurotic woman, and at the time I saw her she had a horrible fear of death from diabetes, as she had been reading some "literature" on the subject. The examination showed a perfectly normal woman in every way; only the merest trace of sugar and no acetone was found in the urine. She was told that if she dieted and took good care of herself she could probably have a normal delivery. However, owing to the intensely nervous nature of the woman, she refused to diet, ate practically what she pleased, including the richest pastry containing sugar. On February 11, 1916, she appeared at my office,

Admission No. 8543 4

MICHAEL REESE HOSPITAL

Name MRS. E. C. F. Ward..... Room. 303

SPECIAL DIABETIC DIET CHART

	FEB 17	18	19	20	21	22	23	24	25	26	27	28	29
MEAT										50	75	75	75
EGGS	4	1/2											
VEGETABLES 5%	200	100							50	100	100	150	150
BREAD					30	40	40	25	40	40	50	50	50
BUTTER										25	25	25	25
CREAM	30				25	25	25	25	25	25	25	25	25
CHEESE							50	50	30	50	25	25	25
BACON	15	10								10	20		10
NUTS									20	20	20	20	20
OLIVES									10	10	10	10	10
MILK			60	1200	1350	900	1000	1000	1000	1000	1000	900	900
BRANDY	50	30											
WINE				240	300	250	250	250	150	60	0		
GLUCOSE			20										
LEVULOSE				6	44	25	4						
PROTEIN	28	5.1	2	40	49						77		
FAT	34	7.7	2	40	51						100		
CARBOHYDR	7	3	24	86	153						81		
CALORIES	620	210	120	1090	1430						1550		
URINE													
AMOUNT	Plain	–	4000	1620	2	2100	1800	1500	2240	2760		1860	2130	2410		2100	1740	1920		1500
SPEC. GRAV	1032	16	09	18	21	21	21	21	12	08		12	09	12		12	10	10		17
REACTION	ac	ac	ac	ac	ac	ac	ac	ac	ac	ac		ac	ac	ac		ac	ac	ac		ac
ALBUMEN	0	0	0.4	0	+	+	+	+	+	0		+	0	0		+	+	+		+
SUGAR %	++	++	0.4	Trace	0	1.6	2.0	1.4	0.7	0.3		tr	0	0		0	0	0		0
SUGAR GMS			16		0	33.6	36.	30.6	15.7	8.28			0	0		0	0	0		0
ACETONE	+	+	+	+++	++	++	++	+	+	0		0	0	0		0	0	0		0
DIACETIC AC	0	0	0	44	+	+	+	0	0	0		0	0	0		0	0	0		0
AMMONIA			2.45	1.52		2.5	2.41	1.97												
BLOOD SUGAR			0.136						0.12											

Fig. 6.—Plus (+) mark signifies that the particular food was given, but was not measured. Except when following "eggs" the numbers refer to grams or cubic centimeters (30 grams figured as 1 ounce; 30 cubic centimeters as 1 fluidounce). The figures in the "eggs" column refer to *number* of eggs, not to weight. The food values in protein, fat, carbohydrate, and calories are approximate computations from published analyses. In giving the specific gravity of the urine the two last figures only are written. The amounts in the ammonia column are actual grams of ammonia per day, as measured by the simple formalin titration.

looking and feeling tired and worn out, with her urine containing 1.5 per cent. sugar and a trace of acetone. She was advised to enter the hospital immediately, but refused to do so. She went to her home and there followed a period of sleeplessness associated with lassitude, nervous irritability, headache, some nausea, increased thirst and increased urination, until the 16th of February, when she finally entered the hospital. Inasmuch as sugar, acetone, and symptoms of acidosis had appeared on a diet entirely too rich in carbohydrates, it was reasoned that she should react well to starvation, and she was immediately placed on a vegetable régime (Fig. 6). Her symptoms gradually grew worse, the headache and nausea became more intense, vomiting appeared, and breathing simulating Kussmaul's air-hunger appeared, with a decided odor of acetone to the breath. The urine showed a gradual increase in the amount of acetone, with a tremendous increase in intensity of acetone and diacetic acid reactions on the third day at the hospital, although the sugar had by this time completely disappeared. On the third day the ammonia excretion was 2.5 grams and the blood-sugar at 3.30 P. M. was 0.136 per cent.—a slight increase for this time of day. Between the second and third days the symptoms of acidosis had increased to such an alarming extent that Dr. DeLee decided to empty the uterus in as rapid a manner as possible. Therefore on the morning of the 19th an abdominal Cesarean section was performed under local anesthesia which had been preceded by a hypodermic injection of morphin. The patient stood the operation extremely well. The baby was delivered, but died in a few hours. Immediately after operation and for forty-eight hours thereafter the condition of acidosis persisted, with a marked amount of diacetic acid in the urine. The patient's room smelled like a fruit orchard. Headache, nausea, and vomiting were constantly present. Immediately after the operation an attempt to combat the acidosis was made. She was first placed on a straight milk-and-water diet, with 2 grams of whisky every three hours, sodium bicarbonate constantly in the water, and rectal injection of 4 per cent. solution of sodium carbonate. However, she could not retain the alkali enemata and her stomach rebelled at the so-

dium bicarbonate by mouth. On the second day levulose was added to her milk, water, and alcohol, and on the third day the symptoms and urinary signs began to clear up. During this period and for several days afterward no attention was paid to the sugar content of the urine and every effort made to combat the acidosis. As just stated, it began to clear up on the third day after operation, and gradually the patient's symptoms disappeared and in a few days acetone was absent from the urine.

When this stage was reached it was easy by proper reduction of carbohydrate in the diet to reduce the sugar in the urine, and within a week there was no sugar or acetone present. The wound healed completely, and the patient left the hospital in perfect health, eating a small amount of carbohydrate, and with the urine completely cleared of both sugar and acetone.

This case brings up for discussion two points in connection with diabetes. The first is the question of pregnancy in diabetes. As is well known, a certain number of pregnant women show, especially toward the end of their pregnancy, dextrose in the urine. This has been more or less of a puzzle until recently, when more careful and more intensive study by means of better laboratory technic has shown that most of these cases belong to the group known as *renal diabetes;* that is, there is no increase in the blood-sugar, such as is always present in true diabetes, and the trace of sugar in the urine remains practically constant regardless of the amount of carbohydrate intake. The work of Frank and others has proved pretty clearly the non-diabetic nature of these late glycosurias in pregnancy.

On the other hand, the development of pregnancy in a diabetic is fraught with considerable danger, according to the opinion of most men who have seen such cases. Not only is this combination dangerous to the life of the patient, but it is very likely to result in the birth of a dead child. Definite conclusions on this subject cannot be drawn because it has been difficult to separate the actual effects of the diabetes from the effects of the treatment that had or had not been instituted. In other words, as is so often the case in medical matters, our conceptions have been based on traditional prejudices. Joslin has been interested

in this subject, and has recently reported a series of cases which at least give more hope to the pregnant diabetic than has hitherto been offered. His results are based on the conception that conclusions are impossible until the pregnant diabetic is treated and studied under the most perfect conditions; if the diabetic receives the benefit of the best medical care then the dangers of pregnancy are not so great. If, on the other hand, the diabetic is simply treated like a pregnant woman, then the effects of diabetes on pregnancy, or the effect of pregnancy on diabetes, cannot be definitely ascertained.

In our case we were dealing with an apparently mild form of diabetes, but, on the other hand, the woman absolutely refused treatment. It is, therefore, obviously unfair to draw any conclusions whatsoever from this case except to say that the pregnancy acting on an untreated diabetes produced an extremely severe acidosis.

This brings us to the second point of our discussion. In our discussion of diabetes and surgery we spoke of the value of preparation as a safeguard against acidosis, leaving the question of the treatment of acidosis until now. It is certainly far easier to prevent than to treat diabetic coma, and when coma has arisen treatment is extremely difficult. The condition of our patient for four days was precarious; in fact, it was so severe that we very much doubted recovery. Whether our treatment by means of milk and alcohol, as recommended by Von Noorden, with finally the addition of levulose, was of real value cannot be determined, but such treatment is indicated in most cases of coma. Some patients, however, react better to complete starvation, yet starvation would have been contra-indicated in this case, in which seemingly the acidosis followed practical starvation. The use of alcohol has a double advantage. In the first place, it is very easily burnt by most diabetics and, therefore, acts as a source of energy. In the second place, in the burning of alcohol the body has one of the best means of defense against acidosis.

The use of alkali therapy in this case was limited simply on account of the inability of the patient to retain sodium bicarbonate either by mouth or rectum. Ordinarily, we would have

pushed sodium bicarbonate or sodium citrate to the extent of rendering the urine alkaline if possible. We might even have given an intravenous injection of sodium bicarbonate, but we held off simply because we did not care to use all our ammunition in one shot. It seems to me that intravenous alkali therapy has two definite indications—first, as an urgent immediate method of getting alkali in the body; and, second, when the use of alkali per mouth and rectum is not sufficient to render the urine alkaline.

Just one word more. Joslin has stated that in his opinion, with the newer methods of treatment, diabetic coma will rarely be seen. The aim of diabetic therapy is the prevention of coma, and its incidence in well-treated cases is becoming increasingly rare. It is, perhaps, well to recognize that coma may ensue not only from diminished carbohydrate content of food, but is especially liable to occur following a "carbohydrate spree." Overfeeding is always to be avoided, and a treated case who *puts one over* on the doctor is very likely to endanger his own life.

CLINIC OF DR. M. MILTON PORTIS

COOK COUNTY HOSPITAL

VOMITING

Analysis of the Causes of Vomiting. Vomiting is Only a Symptom and Not a Disease. Differentiation from Rumination and Regurgitation.

The Mechanism of Vomiting. Special Types are Nervous Vomiting and Cyclic Vomiting.

Treatment and Management.

CHART

VOMITING, in the strictest sense, implies the expulsion from the stomach into the mouth and from the mouth, of part or all of the stomach contents.

The causes of vomiting fall into two groups—

(1) Those acting directly on the vomiting center:

Opium.
Apomorphin.
Tobacco.
Anesthetics.
Uremia.
Diabetic coma.
Onset of acute infectious diseases.

(2) Those acting indirectly or reflexly on the vomiting center: These may be grouped under—

(*a*) Causes acting from the stomach:

Acute gastritis due to irritating foods or liquids.
Poisons, acute infectious diseases.
Chronic gastritis.
Ulcer.
Carcinoma.
Pyloric obstruction.

(*b*) Causes acting from other viscera:

Intestinal obstruction.
Appendicitis.
Gall-bladder disease.
Worms.
Acute pancreatitis.
Floating kidney.
Renal colic.
Uterine displacement and tumors.
Adnexal disease, especially ovarian tumors and adhesions.
Pregnancy.
Hypertrophy of the prostate.

(*c*) Causes acting from the central nervous system:

Offensive odors, tastes, and sights.
Ménière's disease.
Tumors.
Abscesses.
Hemorrhage into the brain and into the meninges.
Parasitic cysts, such as cysticercus.
Meningitis.
Epilepsy.
Migraine.
Sea-sickness.
Hysteria.
Gastric crisis of tabes.

We have today a number of cases, all of which have one symptom in common, and that is vomiting. Let me emphasize that vomiting is only a symptom and not a disease. The patients who have just been examined group themselves as follows:

Case 1.—A patient with all the typical clinical findings of carcinoma of the stomach gives the following history with reference to vomiting: The trouble came on gradually with nausea, belching, and eructations of sour material, followed by vomiting. The vomitus has been profuse. At first the vomitus consisted of food and later it assumed a black color. The black or dark

material resembled coffee-grounds. The vomiting relieved the patient. There has been considerable loss in weight.

CASE 2.—A man seventy-three years old had abdominal discomfort for several weeks before entering the hospital. Associated with the abdominal pain he had irregular attacks of vomiting. He has been constipated ever since the illness began; the bowels do not move even with cathartic measures and move only with an enema. The abdomen has been markedly distended and tympanitic. He retains but a very small amount of liquids or food taken. When the abdominal distention is relieved by an enema he is able to retain some food. The stomach-test shows an absence of free hydrochloric acid, but no blood or Oppler-Boas bacilli. No tumor mass can be felt in the region of the stomach; on the other hand, there is a carcinoma located in the sigmoid region of the colon which is responsible for the tympanites and the vomiting.

CASE 3.—The third patient whom you examined this morning gives a definite ulcer history. I will emphasize particularly the vomiting of which the patient complains. The vomiting is preceded by pain, which comes on two or three hours after meals. The patient feels relieved by vomiting. The vomitus has a highly acid taste, and contains a variable amount of food, but the patient has never seen any blood in it. The laboratory and *x*-ray findings confirm the diagnosis of gastric ulcer.

CASE 4.—A patient with chronic alcoholism has an acute gastritis at this time. He has, in addition, evidence of beginning delirium tremens. The vomiting now is persistent and frequent and contains mucus with some bile. He gives a history for the past few months of vomiting in the morning, the vomitus consisting largely of mucus.

CASE 5 presents the signs of a broken compensation with anasarca. Since the heart has had this disturbance the patient has had indigestion with vomiting.

CASE 6 presents the clinical picture of tabes dorsalis. He gives a history of attacks dating back several years which are typical of the gastric crises seen in tabes. Besides the classical symptom-complex of tabes, the patient has a positive Wassermann and the spinal fluid findings found in syphilis.

CASE 7, a fireman, was suffocated by the inhalation of smoke, and on being resuscitated developed an acute gastritis with repeated vomiting.

All of these cases have had one symptom in common, and that was *vomiting*. Let me emphasize once more that vomiting is only a symptom and not a disease. It is a frequent symptom of a great variety of diseases of the stomach. Vomiting, in the strictest sense, means the return from the stomach into the mouth and from the mouth, of part or all of the stomach contents. The term is used loosely to cover a number of conditions, but I would prefer to use it in the stricter sense, and then use special terms for other manifestations which resemble vomiting, but which really are not, as, for example, *regurgitation of food*. By regurgitation of food we mean the return of food into the mouth and its expulsion from there, the food never having reached the stomach. This is observed most commonly in stricture of the esophagus. Esophageal stricture may be due to an organic disease of the esophagus or it may be a functional disease, such as spasm, particularly cardiospasm, which occurs at the lower end of the esophagus or at the cardiac end of the stomach. In cases of diverticula of the esophagus food is usually returned quickly. It may be delayed for minutes or hours if there is dilatation of the esophagus, and if a diverticulum is present it may be delayed for hours or days. You can determine that the food has not reached the stomach by its neutral or alkaline reaction and by the absence of hydrochloric acid or pepsin. It may, of course, contain saliva, blood, or tumor fragments if the esophageal disturbance is due to a tumor. Let me emphasize too that the regurgitation of this food is usually not accompanied by nausea because the food has never reached the stomach. Even when food is regurgitated violently the patients are not as much disturbed as they are in cases of real vomiting, and after expelling part of the contents may return and take some more food from the table.

The diagnosis of the conditions producing regurgitation depends upon the clinical picture and upon the *x*-ray findings. These conditions are very clearly brought out by the *x*-ray with the opaque meals.

There is another disturbance which must be differentiated from vomiting, and that is known as *rumination*. Some individuals, particularly men who eat too rapidly and irregularly, may regurgitate part of the food into the mouth and then masticate it again and swallow it, just as animals do. Patients who come to us with such a history are typical ruminants. These individuals are frequently of a neurotic type. The diagnosis is very easy, and in the treatment it is necessary to correct both their faulty habits of eating and their neurotic tendency.

Another type of disturbance which must be differentiated from vomiting is one that is associated with the derangement of the ordinary mechanism of swallowing. This condition is seen in bulbar paralysis and also in diphtheric paralysis. The food is not swallowed properly, it is regurgitated quickly, and in diphtheric cases may even be expelled through the nose.

Finally, there occurs in infants a disturbance which can be called vomiting, but which is not real vomiting. When infants regurgitate a part of the milk, there is usually a history of the rapid intake of the milk, and air is swallowed with it. The baby does not know when to stop, and a return of part of the contents of the overfilled stomach along with air bubbles occurs. This is not properly vomiting, for it does not involve the entire mechanism of vomiting which I will now emphasize.

The Mechanism of Vomiting.—In the act of vomiting there are involved the musculature of the stomach, the cardiac and pyloric openings of the stomach, the muscles of the abdominal wall, the diaphragm, and, lastly, the vomiting center in the medulla. During the act of vomiting the cardiac end of the stomach is usually open; the pylorus is frequently closed, although at times it may not be, and some intestinal contents may empty back into the stomach, particularly bile, which may appear in the vomitus. The stomach musculature contracts powerfully, the diaphragm descends as in deep inspiration, the abdominal muscles contract violently, and with this entire mechanism at work, all of it being stimulated by the vomiting center in the medulla, there occurs what is commonly called vomiting. When we have a disturbance of any part of this mechanism, then the

act of vomiting will be incomplete; for example, if the cardiac end of the stomach remains closed, as occurs in cardiospasm, then in spite of the patient's violent efforts at vomiting no vomitus appears. This is called *retching*. The patient goes through the mechanism of vomiting, and yet is not able to vomit. This same act of retching may be due to other defects in the vomiting mechanism.

Causes of Vomiting.—In the consideration of the various causes that may induce vomiting it is best to group them into two large classes:

First.—The causes acting directly on the vomiting center. In this group is apomorphin, a drug which acts directly on the vomiting center, and you are all familiar with the vomiting which can be induced by an injection of apomorphin. Tobacco may act similarly; likewise anesthetics, although in the case of anesthetics one must differentiate the local irritating action, particularly if some is swallowed. Uremia, due to the various renal disturbances, diabetic coma, and the onset of many acute infectious diseases, particularly in children, may exert a toxic action on the vomiting center and induce vomiting.

Second.—The causes acting indirectly or reflexly on the vomiting center. This is by far the larger group and can be subdivided into causes acting—

(*a*) From the stomach.

(*b*) From the various viscera.

(*c*) From the central nervous system.

(*a*) *Causes Acting from the Stomach.*—In this group acute gastritis heads the list. Improper food or liquid which irritates the stomach may produce an acute gastritis which will usually be accompanied by vomiting. It is nature's effort to rid itself of the irritant. I could enumerate in this group a great many causes of acute gastritis, but for our purposes this general statement will suffice. In chronic gastritis we have to consider the various causes that may induce acute gastritis both of infectious and non-infectious origin, and, in addition, the various causes of passive congestion. The latter is commonly due to a heart lesion with broken compensation, as illustrated by one of the cases that

was presented today. However, the passive congestion may be due to any cause which interferes with the return circulation from the stomach. A common clinical example is atrophic cirrhosis. Any of the causes of acute gastritis kept up for a long time will induce a chronic gastritis. Alcoholism is a common example, as was illustrated by another of the patients examined today. In alcoholism the patients frequently have morning vomiting, which consists of a large amount of mucus that has accumulated in the stomach during the night and also some that has been swallowed from the nasopharynx. Ulcer of the stomach and carcinoma, both of which have been illustrated by cases today, frequently are associated with vomiting, particularly if the lesion is located in the region of the pylorus. Obstruction of the pylorus, whether due to a benign or malignant cause, will be associated with vomiting, not only because of the chronic gastritis which is found with the pyloric obstruction, but for mechanical reasons. The cases with repeated constant vomiting over long periods are usually due to this cause.

(*b*) *Causes Acting from Various Viscera.*—This group comprises a large number of clinical conditions which may cause vomiting. It is well to remember that any irritation or inflammation in the peritoneum may induce vomiting. For example, an acute appendicitis is frequently associated in its early manifestations with vomiting. Acute pancreatitis may have vomiting as one of its prominent symptoms. Gall-bladder disease, especially gall-stones, and particularly gall-stone colic, is usually associated with vomiting. Worms of the various types so commonly seen in children and also occurring in adults may produce vomiting. In children they are not an uncommon cause of many obscure cases of vomiting. Intestinal obstruction due to various causes, just as in pyloric obstruction, is associated with vomiting, which is one of the diagnostic signs of the trouble.

Disease of the kidney, aside from the causes acting directly on the vomiting center mentioned under the heading of uremia, may produce vomiting. When the kidney is movable, particularly if it is of the floating type, vomiting may occur. I have a patient under observation in whom vomiting occurs when the

kidney falls from its normal location and causes pressure on the stomach and the duodenum. The vomiting continues until the kidney is replaced. After eliminating every other cause in this patient, I finally had a kidney pad made which the patient wears, and as long as she wears this pad she does not get any of her stomach disturbances. I mention this case, but do not want to emphasize it, for a great many patients with movable kidneys are thought to have enough other clinical disturbances to justify fixation of the kidney. That, however, is not good practice today, for most of the symptoms continue after fixation. Kidney stone producing renal colic may induce vomiting, just as all other types of colic may.

Pelvic disease in women, such as uterine displacement, uterine tumors, adnexal adhesions, and ovarian tumors may reflexly be the origin of vomiting. Pregnancy is a common cause, particularly the morning sickness, and it has probably a double explanation. It may cause vomiting reflexly or, according to some authorities, toxic substances in the circulation may act directly on the vomiting center. In the male a hypertrophied prostate is occasionally the cause of vomiting. I have had patients whose vomiting was relieved after the enlarged prostate was removed. Here, again, there may be a toxic origin as well as a reflex one, for so many of the cases of hypertrophied prostate are associated with urinary disturbances.

(*c*) *Causes Acting Reflexly from the Central Nervous System.*— This is a large group. There are many patients in whom an offensive odor, an offensive taste or sight will induce vomiting. They may learn to control this trouble, but in many cases it persists. Various tumors of the brain, particularly cerebellar tumors, are associated with vomiting. This vomiting, as a rule, is not preceded by nausea, may have no relation to the taking of food, and it may be of the projectile type. Abscess of the brain, hemorrhage into the brain or into the meninges, coma, parasitic cysts, especially cysticercus, all may be the cause of vomiting. Other diseases, such as meningitis and epilepsy, are frequently associated with vomiting. Tabes, with its manifestations of gastric crises, is a common cause. Such a patient was shown at

the clinic today and also one at the previous clinic. These cases are quite common and usually are not diagnosed. Gastric crises so frequently simulate organic abdominal disease that these patients have been operated on for conditions which were purely of tabetic origin. Hysteria, which masks and imitates almost any disease, may give a typical gastric picture with vomiting which simulates an organic disease of the stomach. Migraine is a disease which may cause vomiting and which has received considerable study. We do not understand its pathology definitely, and yet, as in the case of epilepsy, we realize that there is in many cases some brain disturbance associated with the clinical picture.

Sea-sickness is another disturbance associated with vomiting which is still to be explained. It is most likely a reflex disturbance, but the nervous mechanism involved is still obscure. We might mention a great many other uncommon conditions, but we have already named the commonest causes of vomiting.

The time that vomiting occurs, during the day or during the night, is of importance. The time element in relation to the taking of food is of great value in the diagnosis. The relation of vomiting to the ingestion of various kinds of foods or liquids will help to determine at times the different clinical causes of vomiting. It is always important to associate with vomiting the question of nausea, and to realize that when nausea is present the cause usually lies below the diaphragm, and when there is no nausea present it is usually above the diaphragm, and may be a cause acting directly on the vomiting center.

Again, it is important to emphasize the question of pain in relation to vomiting. We will go into this subject in greater detail in the later clinics, but, in passing, let me emphasize that when the pain is relieved by vomiting, usually the origin of that pain is in the stomach.

A study of the vomitus is of great value in the diagnosis. I have already emphasized the character of the vomitus when the food has not reached the stomach. It is then called regurgitated material, as contrasted with the vomitus proper, which implies that the food or material has been in the stomach.

We will consider in detail the gross chemical and microscopic findings of the vomitus in our other clinics when we study the gastric contents in various diseases. Be alert for the odor of vomitus, particularly fecal vomitus, for it has great significance in intestinal obstruction.

There are several types of vomiting which do not classify well in this grouping which I will consider separately. One of the most interesting and at the same time important types of vomiting is the so-called *nervous vomiting*. This often will puzzle you. It usually occurs in nervous individuals; it is more common in girls, who often show the stigmata of hysteria. The vomiting comes on frequently without nausea; it may occur during a meal, and the patient will return to the table and continue to eat without much discomfort. In many ways it resembles closely the type of vomiting seen in cerebral disease. When you observe that the patient brings up only a part of the meal ingested you should think of nervous vomiting as the cause. The patient may retain the most indigestible food and vomit only the digestible type. The patient may be able to retain only a certain kind of food. For example, simple digestible liquids or foods may be expelled and only the most bizarre indigestible types of food retained. This is sometimes called selective vomiting. Although these patients vomit frequently they do not seem to lose in weight, nor is the general health affected unless the condition persists for months, when some anemia and some loss in weight may occur. This nervous vomiting may be associated with other organic disturbances, but when observed by itself is usually caused by hysteria. The diagnosis will depend upon the definite elimination of other causes, the finding of the stigmata of hysteria, and the peculiar manifestations in the patient and in the vomitus that I have described.

The treatment will be that of the underlying nervous disturbance. It is well to make a gastric analysis and attempt to adjust the food and medication by the findings, but, in addition, these patients must be given psychotherapy and the various therapeutic measures, especially hydrotherapy and mechanotherapy, that are useful even in those cases that have no vomiting.

There is a disturbance called *cyclic vomiting* which is seen in children that deserves special consideration. It occurs in nervous, anemic children and may persist up to the age of puberty. The children are usually poorly nourished. The attacks come on suddenly, as at the onset of an acute infectious disease; usually some temperature is present, the child is languorous, and at times stuporous and vomits frequently. The child is usually constipated, and the urine contains toxic bodies, such as acetone, diacetic acid, indol, and skatol. In these children the metabolism should be corrected by such measures which help the natural development and health of the child. The question of adenoids and tonsillar disease should be carefully gone into. In young boys phimosis may frequently be the source of the trouble. In other cases worms are present, and the stools should be searched for worms or the eggs. Again, it may be a question of improper feeding of the child; the child eats too much carbohydrates, is too fond of candy, or the mother does not use good judgment in selecting the food; at times it is a question of irregular eating. In other cases I feel sure that it is an anaphylactic disturbance, that the child is sensitized to certain proteins, and the ingestion of these will produce the disturbance. I have had several such experiences. One child whom I have under observation had for several years attacks of nervous vomiting. A bismuth *x*-ray study revealed the presence of a megalocolon, called Hirschsprung's disease, and attention to this condition by proper elimination and proper local measures has relieved the youngster from these attacks. I emphasize these things, for usually a careful clinical search will ferret out a condition which directly or indirectly is responsible for these disturbances in the child, and I may say that the same is true of migraine. Careful clinical search will frequently reveal some cause which, when remedied, helps, if it does not cure, the vomiting of migraine.

The treatment of the condition described in the children is, of course, forced elimination, if necessary, gastric lavage, and then as soon as the stomach will tolerate it the use of alkalies to combat the acidosis.

I will omit the consideration of the other causes involved in

vomiting, for they will come up later in the various clinical cases that we will study in detail.

Treatment and Management.—In the treatment of vomiting we must remember that there is no specific remedy. The patient should be advised to go to bed and remain absolutely quiet in the horizontal position, avoiding all motion. Sometimes lying on the right side is helpful.

Give small pieces of ice or sips of iced water or iced seltzer. Iced tea and iced champagne are at times retained when other liquids are not.

Unless diarrhea is present, give a soapsuds enema, and if it does not bring good returns repeat the enema and use glycerin and Epsom salts in the water.

Put hot applications on the abdomen and iced applications to the head. If vomiting keeps up, wash the stomach, using a teaspoonful of sodium bicarbonate to a quart of warm water. Wash the stomach until the water returns clear. The newer small tubes, such as that of Rehfuss, will make the procedure less uncomfortable. To allay thirst give a pint of tap water by the bowel, to be retained, and repeat it if necessary. Later, if vomiting persists, a nutrient enema may be necessary.

A great many drugs have been advised, but in my experience most of them are not of great value. Chloroform, 5 drops on sugar, or 10 drops of ether given in the same way, may be tried. Cocain in $\frac{1}{4}$-grain doses is sometimes effectual. Likewise, the tincture of valerian, bromids, chloral hydrate, and nitroglycerin all have been used and should be tried in obstinate cases. Unless an idiosyncrasy is present, the most effectual is a hypodermic of $\frac{1}{8}$ or $\frac{1}{4}$ grain of morphin.

In the cases with periodic vomiting it is most important in the intervals to search out the cause and try to remove it. Analyze the case along the lines indicated by the previous discussion and give suitable medical and surgical treatment. Do not forget in the neurasthenic and hysteric cases to employ psychotherapy along with the usual dietetic, hygienic, and hydrotherapeutic measures.

CLINIC OF DR. RALPH C. HAMILL

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

PRESENTATION OF A CASE OF GENERAL PARESIS

Methods of Onset of General Paresis. Analysis of Patient's Mental Changes. Importance of Reflexes and Sensory Disturbances.

April 10, 1916.

I WILL show this man to you first and go into the history later because I did a lumbar puncture on him this morning and want him to get back to bed as soon as possible.

Examination shows that the pupils are small and the reaction is very sluggish to both light and accommodation. There is a little tremor about the mouth as he talks, but it is very slight. The plantar reflexes are very brisk, and sometimes there is a tendency for the whole foot to go up, the toe as well, so that, although the toe moves in what would be considered a normal direction, one suspects that the pyramidal tract, either at the cortical cells of origin or somewhere below, is affected slightly. The ankle-jerks are quite brisk, the knee-jerks are rather brisk, and the abdominal reflexes are very active. The cremasteric reflex is peculiar; it is quite brisk on the right side, both when the right thigh and the left thigh are stimulated. Both these stimuli give a very brisk response. The left testicle moves very little to stimulation on the left side. The wrist-jerks, on tapping just below the tuberosity of the radius, are very brisk. The triceps and the biceps reflexes are both very active and equal. There is a little trombone-movement of the tongue, a little in-and-out movement, and at the same time there is some tremor around the lips just above the outer angles of the mouth. There

is a slight ptosis of the left eyelid, about 2 mm. of the iris being exposed above the left pupil, whereas on the right about 4 mm. of iris shows. There is, therefore, just a slight ptosis of the left lid. As he stretches out his hand there is a little fine tremor.

DR. HAMILL (to Patient): How long ago did you feel this extreme nervousness? Did you notice it when you were quite young?

PATIENT: I've felt it for at least twenty years.

DR. HAMILL: And at that time you had those attacks of palpitation?

PATIENT: Yes.

DR. HAMILL (testing pain sense): You can all see that there is considerable reduction of pain sensation in the lower limbs. As we go up toward the thigh we find it reaches to about the middle on the left leg and a little bit higher on the right. If you examine him over a long period of time the sensory disturbances are about as I have shown you. There is a band of analgesia about 3 inches wide that goes around the chest just below the nipples. It is characteristic of this band of analgesia that if you prick gently with sharp stimuli as you approach it he will reply "sharp" up to this area (indicating), and then he will reply "dull." On the other hand, if you start with heavy, sharp stimuli and come up to this area he will respond "sharp" all through the test. In other words, the overlapping of the sensory fibers is great enough so that if you give sufficient stimulus in this analgesic area you will get an appreciation of the point of the pin, but if the stimulus is not a maximum one he loses the perception of pain between these lines. This is the difficult part of a neurologic examination—to bring out these lesser changes—but it is very important in the examination of a case of this kind. The demonstration of root lesions in any cerebral disease is very important. They show that besides there being disease in the brain there is also disease in the cord.

The Achilles tendons are not of normal sensitiveness, and I have to use considerable pressure on the testicle before he complains.

The rest of the neurologic examination is practially negative. He has no distinct Romberg; he stands just about as well with his eyes closed as with them open. In making this test you have the patient stand with the eyes open and his feet together and see how much he sways, and then have him shut the eyes and note the difference, if any exists. I think you can see that there is a little increase in the swaying as he closes his eyes, but it is not at all marked. There are no other signs of ataxia. There is practically no change in the optic nerve. It is perhaps a little bit greyer than normal, but the changes are so slight that we cannot draw any conclusions from them. There is the sensory disturbance in the legs and around the chest. Then there are the very sluggish pupils and his mental condition. These are the clinical findings.

The *spinal fluid*, examined at the County Hospital, where he was on my service last December, showed 16 cells to the cubic millimeter, a positive Nonne, and a 4 plus positive Wassermann. An examination four days later showed 30 cells and a positive Nonne. A lumbar puncture this morning showed about 12 cells and a strongly positive Nonne. The Wassermann test has not yet been completed.

Now as to his *history*. He is forty-one years old. He says that since childhood he has been self-centered and would cry easily. He got into the habit of masturbation when about twelve years old, and increasing dissipation in "women, wine, and song" brought him to a crisis at the age of twenty-one. He inherited some money, and took a doctor with him and traveled for some time, recovering somewhat, but not entirely. He says that the masturbation, which he considered excessive, continued between the ages of twelve and sixteen two or three times a day. He then had intercourse and stopped masturbating. He developed gonorrhea at eighteen; at this time he was drinking very heavily, playing the races, and sporting all night. At about the age of twenty-one he lost interest in everything he had been doing, locked himself up in a room in a hotel in Cincinnati where he was staying, and remained there for a month. He did not want to meet people, hated himself, knew he had been a fool, and had not done

any of the things that he should have. He made up his mind that he was mentally affected and tried to figure out when this had commenced. He was frightened, had attacks of apprehension when his heart would hammer hard; he would break out into a cold perspiration and get into a panic. He cried a good deal at this time, but finally recovered sufficiently to go to work for a while. He could never stick to any one job until seven or eight years ago, when he worked as an insurance agent for one of the big companies here in Chicago. He made a good record and got to be assistant manager, and in 1910 brought in a great deal of insurance, showing that he did good work at that time. But every time he would have an interview with a man he was trying to insure he would come away from the interview feeling absolutely done up. He fatigued very readily.

He says that since the time he was about seventeen he has felt that he would much prefer to speak to people over the telephone than face to face. Recently he has had the feeling that he would much prefer to wear a mask instead of talking to people face to face. He is afraid they can see how worried he is and how lacking in self-control. He says this feeling goes back to the time when he was masturbating excessively, and the lack of self-confidence at that time brought this feeling of dreading to meet people face to face.

I might say in passing that this is essentially the deleterious influence that masturbation has on the young. It is the feeling that they are doing something they know they should not do; knowing it very well, and yet unable to resist the temptation. By so doing the continued and repeated blows to their self-confidence have a great deal to do with bringing on the diffidence, shyness, lack of assurance so frequently seen in the individual in the last half of the second decade. I do not know how overweening the race would be if it did not masturbate, but I fear it would be overpowering. But inasmuch as every boy masturbates at some time or another it undoubtedly does influence the self-confidence of mankind. The whole procedure of masturbation, the thinking about it beforehand, remembering that it's the wrong thing to do, being tempted by it, and finally giving way to it, and then having

the physical and mental reaction that comes after it prompt the feeling of self-criticism and self-abasement. The whole act of masturbation and the feelings that go with it have in them the essence of the lack of self-confidence such as this man expresses. He says he used to feel as if he wanted to walk with his arm over his face to hide it.

The religious impulse first comes to the boy just about the time that he begins to realize that he should not masturbate. He knows he should not do it, and yet he does it, and after doing it he swears that he will not do it again, and yet within a few hours he does it again, and he feels that he must have divine aid to help him out.

This man was an unusually sensitive young fellow. He was an only child and had all the attention of his parents lavished on him. This built up in him a strong realization of his own worth, a feeling of his importance. Then these repeated blows to his self-confidence had a tremendous effect. They made him realize the great discrepancy that existed between the person he really was and the person he wished other people to think him. So he hated to meet people face to face and much preferred to talk to them over the telephone. He has had suicidal thoughts for the last three or four years; he thought he would rather commit suicide than lose his mind, but he has never gone further than just to think of self-destruction except at times when he would keep away from the lake for fear the water might tempt him.

This brings out most of the subjective mental symptoms which he volunteers. As to the symptoms which are due to this disease process, he began to have pains two years ago which he called "rheumatism" or "neuralgia." These occurred mostly in the lower limbs; the pains would last several minutes, disappear, and return in a few minutes. These jabs were sometimes continued for an entire day in one area and the next day a new area might be involved. More often these pains were in the fleshy part of the leg than in the joints, and the area which was involved would eventually become very sore to the touch.

When he was eighteen he had gonorrhea. He does not recall that he has ever had a chancre, but when he was twenty-three he

had some sores in the mouth that were called mucous patches. Shortly after that he developed the drooping of the lid of the left eye. It was very much worse at that time, so bad that he could hardly see out of the eye. He went to Hot Springs and had twenty-four rubs with mercury. The next year he went back and had twenty-four more rubs. He had two subsequent attacks of gonorrhea—whether the same old infection or new ones we cannot say. He also had some bladder symptoms. For a good many years he could go for a long time without urinating, sometimes for twenty-four hours, but two years ago he commenced to lose his urine; he would feel the desire to urinate, but would wet himself before getting to the toilet.

He has always had premature emissions on attempting intercourse, and about two years ago while riding in a street car had an erection and emission without any lascivious thoughts.

There was one thing I brought out while examining him that you may have observed. While I was questioning him I was sticking him in the leg very hard with a pin. This morning he felt jabs like that when I called his attention to them, but when I engaged him in conversation he did not feel them. This is very characteristic of general paresis. If you talk to these patients and get them to thinking about something else you can jab them in the legs without the least manifestation of pain on their part.

Those are the principal things in the history of this man, and it is a rather typical history of paresis on the basis of an unstable individual. He has always been unstable; he gave way to the desires of dissipation when a young boy. His father died when he was sixteen and left him considerable money and he "went the route." He showed this instability when he shut himself up in the hotel in Cincinnati and tried to think of the time when he first had his mental trouble. Doubt of his sanity has persisted ever since. Recently he has had no desire to make his living; he has been married for eight years, but makes no effort to support his wife, and shows no signs whatsoever of acknowledging his duties. He shows these physical characteristics of syphilis of the central nervous system—the sensory disturbances, the pupillary disturb-

ances, the tremor around the mouth, the abnormalities in the spinal fluid. At the present time he presents the picture of a man who is tired out. We call that condition neurasthenia. We are struck by the unwillingness to make any effort, to meet any responsibility. The neurasthenic onset of general paresis is a well-recognized one. He was unstable when a boy and dissipated very badly. We cannot tell whether he would have stopped sowing his wild oats when he was twenty-one or twenty-two if he had not developed syphilis. But at about that age he did develop syphilis, and he has had a diseased central nervous system since that time. He has never had sufficient treatment, and now he is undoubtedly developing paresis.

The **methods of onset of paresis** are the neurasthenic, such as this man typifies; the maniacal, like the man I recently saw who was on his way east from California and suddenly one night woke out of his sleep and began smashing the windows and everything in sight. His friends had had no idea that there was anything the matter with him before that time. Then there is the convulsive mode of onset. A man will be going along, doing his work all right, and then have a convulsion and never be right from then on.

There is no question but that general paresis is due to syphilis. We obtain a history of syphilis in 70 to 80 per cent. of the cases. Then there is a large number of cases in people who, so far as they know, have never had a chancre, but they have had the other signs. This man belongs in this category. He had no definite chancre so far as he knows, but he had the attacks of gonorrhea, one of which may have been an intra-urethral chancre. These cases are frequently called gonorrhea, there being nothing to point to the syphilitic nature of the infection. Many men will confess to a doctor their venereal history and yet deny that they have ever had a chancre, and it is very likely that in these cases one attack of so-called gonorrhea was an intra-urethral chancre.

He has no megalomania; none of the grandiose delusions which we so often see in early paresis. That may be due to the fact that he has always had this feeling of abasement. The megalomania

feelings of paresis I think can be explained on the following basis:

There exists in every man a certain appreciation of himself which, in spite of his failures, of which he has full cognizance, leads him to feel he would rather be himself than change with any one else. Perhaps this is due to a sense of "at homeness" or familiarity with the interior furnishings and decorations of his mind. He becomes accustomed to a certain mental working speed and working ability. In paresis this speed and ability are cut down. The paretic is no longer able to function mentally as he once did. One of the earliest developed mental functions is self-appreciation, as is seen in the unbounded egotism of the child. It is perhaps a remnant of this childish egotism that persists in the adult feeling I just mentioned, of not wanting to change his personality for that of another. This egotism persists long after the organic changes in the brain make critical judgment impossible. It is a direct outgrowth from the instinct of self-preservation and can be thought of as capable of existing in the more simple brain of a child, at least as not requiring the full complexity of the normal adult brain. The power of judgment, however, does require a brain capable of its best work, does require a brain anatomically in good repair.

Now, as a paretic's mental powers fail, as he becomes less and less able to meet the competition of the world, and as his power of self-criticism fails, he feels the necessity of making the exaggerated claims for himself which we call megalomania. This ego of his, which he still cherishes and will not trade, needs value given to it. Also, as the organic changes go on, his mind tends to become more and more like the mind of a child so far as the breadth of its interests are concerned. This losing of the broader interests, the interests acquired through days, weeks, and months of experience, leaves *self* without much distraction. If he can't *be* the strongest, the wisest, or the richest man in the world he can at least have the pleasure of saying he is.

In these megalomanic claims we perhaps see a man's last defense before the final wreck. I had a patient out at Dunning who used to say, "I'm Julius Cæsar, I'm Napoleon, I'm Jesus Christ,

I'm Charlie Wright (his own name). He was an alcoholic and had never amounted to anything, but he wanted to be right up in the front row with the big ones, and his paresis enabled him to have all the elements of pleasure and he was happy in it.

The patient I have shown you today must have intensive treatment. I propose to give him some of the arsenic preparations and load him up with mercury. I shall also advise intraventricular injections of mercurialized serum. We may not be able to stop the paresis, but he may improve markedly.

MANIC-DEPRESSIVE INSANITY OR RECURRENT MELANCHOLIA ON A BASIS OF DYSTHYROIDISM

Periodic Attacks of Nervousness, Depression, and Weakness Accounted for by Disturbed Thyroid Function.

April 24, 1916.

THIS woman, a native of Ireland, fifty-four years of age, came to the hospital on the 16th of April complaining of great weakness, depression, and nervousness. She had ringing in the ears, pain between the shoulders, loss of appetite, and slight constipation. She sleeps only four or five hours at night. The present trouble began fifteen years ago, with attacks which have occurred about once a year. For several mornings she would wake up with palpitation of the heart, a sensation of choking, and weakness all over. After waking for several mornings with these symptoms, which can be ascribed to apprehension, the nervousness, depression, and weakness would gradually develop, lasting for several months, and then would gradually disappear. She would be comparatively well for the remainder of the year, when another attack usually came on. She says these attacks occurred most frequently in the spring.

DR. HAMILL (to Patient): Did the weak feeling with the hard breathing and beating of the heart pass away after you had been up and around for a while.

PATIENT: Yes, but in the evening it would all come back and I would only sleep a couple of hours, and then the nervousness would be just as bad.

DR. HAMILL: With the mere going to sleep she would lose the feeling of safety she had gained during the day, and the apprehensiveness that marked the beginning of the attack would come back. The apprehension not being fulfilled all day long gradually wore off. By supper time she would be in a fairly calm state of mind and for a little while she would be like

herself, as she says, but always with this supposed danger lurking behind her. (To Patient): What time did you usually go to bed?

PATIENT: About 9 o'clock. I would feel all right when I went to bed, but if I slept for even a few hours it would all come back and I got weaker and weaker.

DR. HAMILL: And that would continue through the attack. Would you have the short periods of feeling fairly well all through it?

PATIENT: Yes, I always felt a little better by supper time.

DR. HAMILL: How would you feel when the attack finally passed away?

PATIENT: I would come right back to my natural way again; not very strong, I'm never very strong, but I would be natural again.

DR. HAMILL: On inspection you can observe a little swelling of the right lobe of the thyroid. The patient first noticed this when she was eighteen years old. It increased in size when she passed through the menopause five years ago, and she has noticed that it becomes larger every time she has one of these attacks. After the attack is over it becomes a little smaller again.

There is a faint suggestion of a tremor as she holds the arms out straight in front. There is no Stellwag or other ocular sign of exophthalmic goiter. The pulse-rate averages 76 to 78, the respirations are about 20. The blood-pressure is 108 systolic, 74 diastolic. (To Patient): Do you lose much weight during these attacks?

PATIENT: Yes, I always lose a good deal.

DR. HAMILL: Do you have bad sweats during the attacks?

PATIENT: Oh, yes; that is one of the symptoms of them.

DR. HAMILL: How old were you when you started to have trouble with your teeth?

PATIENT: I was just a young girl.

DR. HAMILL: She has had the ordinary diseases of childhood. The menses ceased five years ago with a very heavy flow. They began at sixteen and were always normal except that at seventeen

they disappeared for ten months. She has been married for thirty-two years. She has had six pregnancies. The first labor was quite difficult, but normal. She nursed all of her children except the first two. Her husband is living and well. Her father died at forty of pneumonia, her mother at sixty of paralysis. She is the youngest of ten children, several of whom died in early life. There is no history of any family disease or nervousness. She taught school for sixteen years and then did housework. She has always had trouble about money matters. She has taken no alcohol for some time and never took medicines.

So here we have the essential parts of her history. The attacks began at about the age of thirty-nine. The small goiter appeared at eighteen. She has had trouble with her teeth since she was sixteen. All the upper teeth have fallen out and all but six or seven in the lower jaw are gone. One after another they have fallen out from decay or abscess at the roots. This makes a fairly consecutive picture and the sequence of events would be considered in this manner:

First, there is the trouble with the teeth. The infectious material from the gums caused an inflammatory process in the thyroid gland, a chronic inflammatory thyroiditis. As a result there was a slight increase in the internal secretion of the thyroid gland. The increase of that secretion accounts for much of the picture which she presents. There is, on the physical side, a fine tremor. Usually there is with hypersecretion of the thyroid also a rapid pulse, staring eyes, and the face of a person who is terrified. This woman does not present the facial appearance of exophthalmic goiter, but she has the mental symptom of fear and the subsequent depression that come from being in a terrified state. So while the picture lacks the exophthalmos, it has the slight goiter, the fine tremor, and the mental state. She has, in addition, hot flashes and heavy sweats.

Diagnosis.—What diagnosis is to be made in this case? She has periodic attacks, about one a year. She has occasionally gone a year or two without an attack, but almost always has one in the spring which lasts for several months. During these attacks she first has the apprehensive spells, then becomes more and

more nervous and depressed. During that period of depression she dreads the tasks that she has to perform and feels that she is not able to do her work properly. She has crying spells and the desire to cry continuously. She has also had thoughts of suicide. These are all symptoms that we see in manic-depressive insanity and in melancholia. Although that is the mental picture, the physical signs point toward the thyroid gland being responsible for this mental state. The presence of the enlarged thyroid gland suggests the depressive periods that are known to occur in connection with exophthalmic goiter rather than a pure psychosis. About six years ago the palpitation became so pronounced that she was thought to have heart disease, and a very well-known physician in Chicago gave her tincture of digitalis for this supposed heart trouble.

I think we can diagnose this condition as recurrent melancholia on the basis of hyperthyroidism, at least on the basis of dysthyroidism. We are not sure whether there is hyperthyroidism or dysthyroidism, but there is certainly a disturbed function of the thyroid gland. The facts that speak for hyperthyroidism are the slight tremor, the sweats, hot flashes, and the enlarged gland. Speaking against it is the low pressure, although that can occur in Basedow's disease, the fact that her pulse-rate is not rapid, and the absence of ocular signs.

Treatment.—What can be done for this patient? In the first place, it is necessary to clean up the infection in the mouth. When that is accomplished we can hope for some improvement in the thyroid gland. If no improvement results, then it is time to think of surgical interference—the ligation of arteries on the right side which supply the thyroid gland, or even the excision of the right lobe and the isthmus. Until we decide that such surgical procedure is indicated we have to treat her symptomatically. We will give her a vegetable and fruit diet, eliminating the proteins as much as possible, because it is known that proteins are harmful in hyperthyroidism. For the depression we can give her a combination of hydrotherapy and medicine. She now gets a hot pack lasting for half an hour three times a day. The sheets and blankets are moistened, and then a tin frame like the top of a tunnel is

put over her. On the under surface of this cover are about a dozen electric lights, which are lighted and left on for about half an hour until she sweats profusely. This has a quieting effect and a very good influence on her state of mind. She feels she is getting better. That is a great help in these cases that are so largely mental. But I am rather doubtful whether that will be sufficient, and I think we shall have the teeth extracted and the thyroid gland treated in some surgical manner.

PERIPHERAL NEURITIS ON A SPECIFIC BASIS

Characteristic Combination of Symptoms. Improvement Under Antispecific Treatment.

THIS patient, an Italian woman, thirty-seven years old, a factory garment-worker, was admitted to the hospital on the 30th of March. She was brought to the hospital in a wheel-chair because of inability to walk, pain over the abdomen, extremities, and thorax. It is difficult to obtain a history because she speaks no English. The pain started six months after a profuse discharge from the vagina. She was operated upon, but did not feel well after returning home. She has had no discharge since then, but has not been able to work and has been in bed most of the time. The abdominal pain is slightly worse after eating. She had the usual diseases of childhood, but was never very sick until two years ago, when she began to have cramp-like pain in the abdomen. This was relieved by medicine, but later returned. For the past six months the pain has been practically constant.

The family history is negative. She has three children living and well out of a total of nine births. Six children died during infancy of unknown causes.

Physical Examination.—We see a poorly nourished person who seems to be in no pain now, but was in considerable pain when she entered the hospital. The calf muscles on either side are tender; the external perineal nerves do not hurt her much. The muscles of the thighs are slightly painful and the muscles of the arms, particularly the left forearm, are slightly tender. All the arm muscles were tender when she first came to the hospital.

Sensory Disturbance.—You have noticed how long she complained after I stimulated the sole of the foot; she responded at once and then there was another complaint of pain. Whenever you see that symptom you must think of peripheral neuritis. It is one of the most striking things that we see in that condition

and is very seldom seen outside of peripheral neuritis. She complains of pain from the passing of the percussion hammer over the sole of the foot. It would cause a slight tickling sensation in a normal person, but no particular pain. I also jabbed her three times with a pin before she complained of pain. The first two jabs she did not feel at all. Up here (indicating) I pricked her good and hard five times before she complained. The combination of those two findings is very characteristic of peripheral neuritis; that is, the hypersensitiveness of the foot sole combined with the hyposensitiveness of the dorsum of the foot to the pin-prick. I can press here with my thumb, not hard enough to hurt a normal individual, for three seconds before she complains of pain. She stands the pain of the pin-prick better than that of the thumb. The response to both deep and superficial stimuli is diminished. We have examined her touch sense in the legs, and while it is somewhat diminished it is not diminished nearly as much as her pain sense.

Reflexes.—There is absolutely no knee-jerk and no ankle reflex on either side. There is no wrist-jerk; the triceps and biceps jerks on either side are absent. Her eyes react fairly well to light.

So on physical examination we find a partial lack of function. She can move her extremities in all directions, but there is considerable ataxia when she attempts to put one foot on the knee of the other leg. The same thing is present in the arms; in attempting to touch her nose with her finger with the eyes closed there is a good deal of uncertainty—she has to feel for the nose. That ataxia is not only in the large movements, but in the finger as well; she holds her index-finger on her thumb to steady it. The cranial nerves are unaffected. She has lost considerable muscular power.

Diagnosis.—The first thing to note is the peculiar combination of sensory disturbances—the hypersensitiveness of the foot soles to weak stimuli, with the hyposensitiveness of the dorsum of the foot to light stimuli, and the muscular tenderness. Along with these characteristic findings there is the marked ataxia, the generalized weakness, the loss of flesh, which belong to

the picture of a peripheral neuritis. There is lacking the vasomotor disturbance, such as we see particularly in alcoholic neuritis, in which the feet become very cyanotic.

The blood-serum showed a strongly positive Wassermann reaction; the spinal fluid gave a negative Wassermann. The urine was negative. The blood shows some anemia—3,376,000 reds, 70 per cent. hemoglobin. This suggests the possibility of pernicious anemia, as it gives a plus 1 color index, but as a rule the subcutaneous tissue would be much better preserved than it is in this patient. Moreover, the combination of symptoms points strongly to the peripheral nerves rather than to the spinal cord itself. She has the lost reflexes and the sensory disturbances which are seen in pernicious anemia, but in her case there is much more peripheral wasting. The diagnosis of syphilis is borne out by the therapeutic result. She is very much better since she has been on mercurial inunctions.

Treatment.—The treatment for this case is the ordinary treatment for syphilis. She is getting 2 drams of mercurial ointment each day, with careful attention to the teeth. She says she now feels much better than she did when she came to the hospital.

A TYPICAL CASE OF BASEDOW'S DISEASE

Physical Manifestations of Apprehension. Tonsillitis the Etiologic Factor.

In contrast to the previous case, in which the symptoms can be considered as the *mental* manifestations of apprehension, if I may be allowed to use the term, is this case of thyroid disease where the *physical* manifestations of apprehension occupy the prominent place. I wish to show the two together in order to emphasize the element of apprehension which seems to be so frequent in cases of thyroid disease.

You will remember that in the previous case the attacks of depression were always preceded by a period of a few days in which awakening was always associated with rather violent attacks of apprehension—cardiac palpitation, smothering sensation, and the feeling that some danger was impending. These are the symptoms of apprehension, and, as I have said, they always preceded the development of the depressional phase of the psychic attack. These mental manifestations, along with the loss of weight during the attack, and the marked sweating, I have attributed to disease of the thyroid gland. In this second case the sweating and change in weight form a connecting link, as does the idea of apprehension. In this case the manifestations of apprehension are very largely physical rather than mental.

As you look at the patient you see her wide, staring eyes, a slightly drawn expression around the mouth, and a quivering now and then of the upper lip. The pulse is running between 115 and 125. It is slightly irregular and the heart sounds are rather loud. This facial expression and the rapid heart-beat have been spoken of as typical of a physical expression of fear or apprehension.

The *history* of this case is as follows: The patient is forty-one years old and has been married for eighteen years. Her father died of rheumatism at the age of sixty-four, her mother of pneumonia at seventy-two. Two brothers are dead of unknown causes. One sister has paralysis, cause unknown. The patient herself has had "tonsillitis" often since the age of six. The tonsils were snipped off with a tonsillotome when she was seventeen, but in spite of this partial removal she has had sore throats off and on ever since. When she was eighteen an enlargement of the thyroid gland was noticed. She says it was very much larger then than it is now. At that time she had shortness of breath and puffiness under the eyes and was very nervous. The symptoms of thyroid disease apparently came and went. When she was twenty-four she "was afraid to walk because her heart beat so hard." In fact, ever since she was eighteen any infection or any severe emotional strain has been sufficient to produce signs of increased trouble with the thyroid gland. Of late she has had a good deal of trouble with her husband, who has dementia paralytica. This trouble has made her so nervous that she has had to stop work. Her husband's dementia paralytica developed from a chancre acquired by him twelve years before his marriage. The patient's blood and spinal fluid are both entirely negative to tests for syphilis. The fact that her thyroid enlargement came several years before her marriage naturally makes it highly improbable that the syphilis has anything to do with the origin of her present trouble.

To return to her symptoms. You see there is this staring of her eyes—in other words, an *exophthalmos*. Stellwag's sign is moderately positive; that is, the upper eyelids do not follow the downward movements of the eyeballs perfectly. Still, there is but little sclera showing above the iris as the eyeballs turn down. The Moebius symptom is negative; that is, she can look at the finger when it approaches to within a couple of inches of her nose, holding the eyes turned in, with marked strabismus. The ocular movements are all normal, the pupils are normal, the optic nerves are normal, although the physiologic cup is not well marked in either fundus, and there is a crescent of the

sclera showing through the choroid around the temporal border of the right disk. The facial nerves are normal in function, the reflexes are brisk, but equal; she shows a fine tremor of the outstretched hands as well as the one you notice about her mouth as she protrudes her tongue. The tongue is also slightly tremulous.

The apex of the heart is in the midclavicular line in the fourth interspace, or perhaps behind the fifth rib. The right border of the heart reaches midsternum.

I would call your attention to the perspiration you see dripping from the axilla. (To Patient): Does any little excitement, such as talking to you in this way, cause you to perspire as you are now doing?

PATIENT: Yes, sir.

DR. HAMILL: You will also notice how flushed the skin is around the neck and over the upper chest. There seems to be a marked dermographia. The swelling of the thyroid gland is confined to the isthmus and the right lobe. In the neck you can see the marked palpitation of the carotids. The blood-pressure is 150 systolic, 78 diastolic. Her temperature is normal; there is no marked variation from normal in the blood-count. Both tonsils are enlarged, the right a little more than the left, and from the right I am able to express a drop of pus. The lymph-glands in the neck are not markedly enlarged.

This, then, is a more or less typical case of exophthalmic goiter, or Basedow's disease. The two factors of interest about it are its origin and its comparison with the previous case. I have already spoken of this comparison. My last comments on the results of the examination of the patient were relative to the tonsils, and, if you will remember, attacks of tonsillitis began at the age of six years and have continued, in spite of the partial removal of the tonsils, off and on ever since.

Can we assume a causal relationship between the tonsillar condition and the thyroid disease with its manifestations? I believe we can. I think we have to assume that the mechanism is somewhat like this: The patient developed early in life an infectious focus in the tonsils. They either became permanently

infected with a micro-organism whose virulence varied from time to time, or else the tonsils became a *locus minoris resistentiæ*, a place of lowered resistance to micro-organisms such as we are constantly taking into our mouths, and became reinfected at frequent intervals. Whichever theory is the true one, the fact remains that once or twice a year she developed attacks of tonsillitis. For many years we have recognized such attacks of tonsillitis as active factors in cases of acute inflammatory rheumatism and chorea. Proofs are accumulating to show that many other conditions previously spoken of as idiopathic are, in reality, due to the introduction of pathogenic micro-organisms through the tonsils. Exophthalmic goiter is one of these conditions.

In this case I think the causal relationship, is beyond question, first, the tonsillitis, then the thyroiditis. The latter has persisted for a good many years. Consequently, there is undoubtedly a hypertrophic condition in the thyroid. In other words, organic changes have taken place, and although the present symptoms may be due merely to an increased function of the gland, the mere removal of the causative factor of the thyroiditis may not be sufficient to cure the disease.

Treatment.—Without question, the first step to be taken is the removal of the tonsils. Then, with rest in bed, with 10 grains of sodium bromid three times a day, a restricted protein diet, and care of the eliminations, we will note whether improvement takes place, and wait before deciding whether the thyroid should be operated upon or not. While she is in bed I propose to give her some intramuscular injections of bichlorid of mercury in order to bring out a latent Wassermann, in case she did acquire syphilis from her husband.

CLINIC OF DR. CHARLES LOUIS MIX

MERCY HOSPITAL

STAPHYLOCOCCIC OSTEOMYELITIS DEVELOPING AS A RESULT OF INFECTION FROM TEETH

March 28, 1916.

FATHER C. is forty-one years of age. He entered the hospital in March, 1915, complaining of pain in the left ankle and in the right wrist. There was also some discharge from the left ankle which had been opened to let out some pus before he came here.

The family history is negative as to tuberculosis, carcinoma, and rheumatism.

The patient had scarlet fever, measles, and mumps in childhood. Two years ago a small fatty tumor was removed from the right elbow. Aside from this his health has always been good until the present trouble which began in November, 1914, with severe pain in the left side of the lower jaw. This pain was thought to be neuralgia and, as a matter of fact, it was. He went to a dentist, who discovered an infected wisdom tooth on the left side of the lower jaw, and he extracted it. The following day the pain appeared on the right side of the jaw. He again went to his dentist, who found nothing wrong after cleaning the teeth, and the pain was unexplained. No skiagram was taken at this time by the dentist, yet I am informed that dentists cannot tell exactly regarding the condition of the roots without the aid of skiagrams. The pain was so severe in the right side of his face that he could not sleep or eat, so he was forced to go to the dentist for the second time two days after his first visit. On this occasion his dentist removed two

fillings, but, finding nothing abnormal in the teeth, put the fillings back. After a lapse of about two weeks, the pain continuing all the time, the patient consulted a physician who gave him medicine to relieve the neuralgia, and insisted that the dentist again examine the teeth carefully. At this time a tooth was found on the right side which was more sensitive than the others. It was drilled, but nothing was found in it, and the tooth was filled. After this had been done a skiagram was taken of all the teeth, and a tooth on the left side was thought to be diseased. It was opened, but nothing was found in it, and it was also filled.

All this time, up to January, 1915, he had continuous pain in the right side of the jaw and some loss of weight. In January, 1915, the pain left the face and began to affect the back of the neck, suggesting the possibility of an involvement of some of the articular facets of the vertebræ by an inflammatory process, possibly infectious and distinct from the neuralgic process which was evidently toxic. Following the apparent joint pain in the back of the neck he also had pain in the right arm and the right foot. He describes the pain as aching in character, like a toothache. During May and June, 1915, he took some massage and chiropractic treatments without relief. Indeed, the pain became so severe that he could neither sleep nor walk. He went to northern Minnesota during the summer, but grew worse. He then went to northern Wisconsin where he was examined carefully by a physician, who, for the first time, discovered that he was suffering from an advanced case of pyorrhea. He was put in a hospital and given injections of emetin and local treatment to the gums, and left in about three weeks somewhat improved.

On the day the patient left the hospital in order to come to Chicago for further study into his case he fell and suffered a right Colles' fracture. This necessitated his return to the hospital, where he remained for two weeks longer. He then came to Chicago, and when I saw him the question that had to be settled was whether the supposed neuralgia and the other symptoms he was complaining of were due to his teeth or to a focus of infection in some other location.

After examining him very carefully as to his tonsils, head sinuses, gall-bladder, appendix, and prostate gland we were certain that his teeth were responsible for the whole of his condition, and referred him to a dentist for treatment. The hard tartar on the teeth was scaled off and they were properly treated. Within three weeks' time the pain had disappeared from the right foot and the pain in the left foot was very much lessened. He received daily treatment for his teeth with the application of emetin locally about the gums, and the pyorrhea was materially helped. He returned to his home in November, 1915, in very much better condition after a thorough cleansing of his teeth.

The patient was not cured, however. In December, 1915, after he had been home for about one month, pain again started up in his jaw and in both ankles, and he returned to the city to take some more treatments from the same dentist. These were successful, and before the holidays he went home feeling very much better.

In February, 1916, he again had pain about the jaw, this time in the upper jaw. The pain also returned in both of his ankles. On this occasion he came again under our care, and we had skiagrams made of his teeth, finding a new pocket of pus and some decay in a tooth on the right side. This was treated by the dentist and the pain in the jaw disappeared, but the pain in the left ankle remained.

It will be remembered that the patient had a Colles' fracture of the right wrist in the fall. This fracture had apparently healed up during midwinter, but in February heat, redness, and swelling reappeared in the wrist at the site of the fracture. Moreover, the left ankle, which had swollen up during his absence, had been incised by a physician and pus was found. Skiagrams were accordingly made of the left ankle and of the right wrist. The skiagram of the left ankle showed a suppuration just beneath the periosteum at the end of the fibula just below the external malleolus down as far as the tip. The skiagram of the right wrist showed a focus of pus in the distal fragment of the radius close to its articulation with the wrist

bones. The skiagram shows this pocket as a slightly more transparent area in the bone.

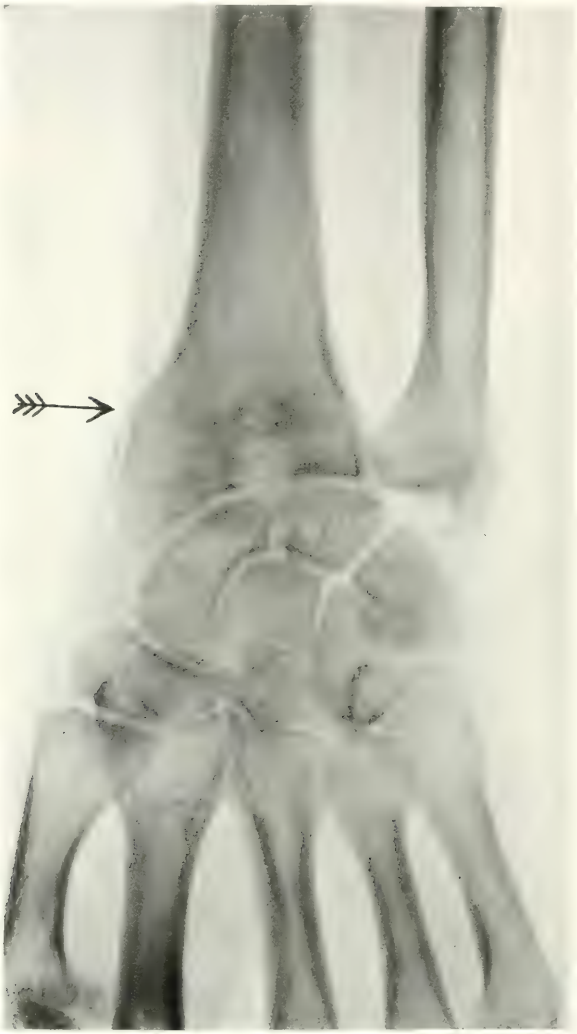

Fig. 7.—Staphylococcic osteomyelitis. The arrow points to a metastatic focus of infection in the radius.

The interpretation which we placed upon these plates was that the focus in the left malleolus, which had been opened at

his home by a physician, was an osteomyelitic focus affecting the lower end of the fibula. Our x-ray showed it to be a subperiosteal affair. The focus in the right wrist was looked upon as an osteomyelitic process occurring in the distal fragment. The skiagram (exhibiting skiagram in shadow-box) shows at this point where the Colles' fracture took place, and you will notice the line of fracture. The dark spot in the plate, which will show light in the reprint, represents a small collection of pus in the distal fragment close to the articulation with the bones of the wrist. It is evident from these two plates that micro-organisms from the abscessed teeth got into the blood, producing these two osteomyelitic foci. You will notice that the tip of the left fibula is indistinct, while the remainder of the edge is sharply defined.

Dr. John Golden, in the absence of Dr. Murphy, opened up the left ankle and cureted it. It is now almost well. The focus in the right wrist was opened, the bone cureted, and drained. We expect it to get well. A culture was taken and a twenty-four-hour growth from the wrist showed a staphylococcus. The reason that the secondary infection in his case is so mild is that it is a staphylococcic infection. Had it been a streptococcic infection it would have been far more virulent.

You will notice that the patient does not look well, that he is somewhat pale, although well nourished. He has the appearance which one notices in cases showing a mild degree of sepsis. Attention must also be called to the fact that when he entered the hospital he showed no increased leukocyte count—6800. The hemoglobin, however, was reduced to 70 per cent.

The outcome of this case is going to be a complete cure. The last of the pus has been taken out. The dentist has done excellent work, and when these two foci in the bone are gotten rid of he will be perfectly well.

Diagnosis.—The diagnosis is, therefore, staphylococcic osteomyelitis secondary to a disturbance on the part of the teeth. It is very easy to fall into pitfalls in dealing with these cases. Here is an individual who at his home for sixteen months past has been regarded as a case of primary neuralgia with an under-

lying rheumatic taint. Yet the cause of his neuralgia and his apparent rheumatism was due to foci of infection in his teeth. Had this case been seen a few years ago he would have been called a classic type of chronic articular rheumatism, but in the light of recent discoveries we are now enabled to say that both the bacteria and their toxins were absorbed into his blood from the foci about his teeth. This is evident from the skiagrams and from the finding of pus at the time of operation.

A great deal has appeared in medical literature in the last few years concerning the teeth as foci of infection. The teeth probably act as foci in a large number of cases, although probably not to the extent that certain enthusiasts would have us believe. Some have gone to extremes in their accusations of the teeth as foci of infection, and large numbers have been needlessly extracted. Nevertheless, there has been much of value in the statements of extremists, because they have succeeded in bringing to our attention the teeth as possible foci of infection in a large number of cases hitherto imperfectly diagnosticated as regards their etiology.

Pain in the jaw was one of the earliest manifestations of disease of the teeth in this case. The pain may be on the same side as the infected tooth, but it is not invariably the case, because toxins may be absorbed and affect nerves in various parts of the body. Not infrequently when a tooth of the lower jaw is involved the pain is referred by the patient to the upper jaw. The reason for this is that the great auricular nerve is given off from the inferior maxillary nerve in association with the inferior dental. Disturbance in the supply of the inferior dental may, therefore, be referred by the patient to the territory of supply of the auricularis magnus. Thus, a pain in the cheek and temple may actually be due to a diseased tooth in the lower jaw.

No examination is complete in such cases of facial neuralgia which does not investigate the territory of supply of Meckel's ganglion. You will remember that the nerves which originate from Meckel's ganglion supply a large amount of mucous membrane in the head. It is, therefore, possible to have very in-

tense neuralgia from disturbance of the various sphenoid, ethmoid, and superior maxillary sinuses. Furthermore, the tonsils are frequently a source of infection. Therefore one must always examine thoroughly the teeth, sinuses, and tonsils in cases of this type.

When you examine the teeth be sure to have skiagrams made of all of them. You can have three plates made of the upper and three of the lower jaw, so that you can have every tooth radiographed.

Besides the facial neuralgia in this case and the foci in the fibula and radius, there were also manifestations in other of his joints, and in the articulations of the vertebræ in his neck. Remember that neuralgias associated with such pains mean almost invariably the absorption of toxins from some particular focus, and that the neuralgia often originates from the teeth. Indeed, these should be examined first, because they are more often the real culprits than the sinuses.

This patient will recover completely after having suffered for sixteen months. The foci of infection have now all been done away with, and he will be cured. I asked him to let me present his case because it is an excellent proof that a mild degree of osteomyelitis may result from infection of the teeth.

NOTE.—Father C. left the hospital in April, 1916, in excellent condition and with the look of health replacing his pallor. Today (May 23, 1916) we have heard from him. He states that he is doing his full and usual work for the first time in over a year.

PRESENTATION OF A CASE OF PARAPHASIA

Type of Speech Disturbance Known as Wernicke's Conduction Aphasia. Localization of Cerebral Centers and Association Pathways. Etiologic Role of Thrombosis and Embolism.

March 28, 1916.

THIS rather unusual case is that of a physician, an ophthalmologist, who entered the hospital on March 15th. On entrance his pulse was 108, his temperature 97.8° F. The pulse ranged from 100 to 108 until 8 P. M., when the temperature began to rise, being 99° F. at 8 o'clock and 100° F. at midnight. His pulse meanwhile had reached 120. When he was brought to the hospital he was talking irrationally and at random. There was much emotional disturbance and restlessness, and at first he refused to have his temperature taken.

On the following day he had a normal temperature at 6 A. M. (these are all axillary temperatures), but at 1 P. M. it was 100.8° F. and at midnight 100° F. The pulse ranged from 88 to 122. During the day he talked incessantly and was very restless. At night he was wakeful, dyspneic, and constantly trying to get out of bed. During the night of the 16th he rested a little better and more quietly than the night of the 15th.

On March 17th the temperature was only 99° F. with a maximum pulse of 80. On the 18th the maximum temperature was 100° F., the maximum pulse 132 at midnight, and his respiratory rate increased. On the 19th the temperature was more nearly normal, ranging from 98.4° to 100° F., with the pulse varying from 88 to 100. He was more quiet and did not attempt to get out of bed as much as he previously had done. On the 20th the temperature remained normal until midnight. On the 21st subnormal temperatures began to appear. At 4 P. M. on that day the temperature was 96° F., and during that day his pulse varied from 70 to 92, his condition in general being much improved.

On the 22d his temperature ranged from 97° to 97.4° F. and he was better than he had been at any time.

During all this time, however, he had involuntary urination, but no involuntary defecation. On March 26th his condition changed rapidly for the worse because of the development of pain in his left leg. The cause of this pain and the rapid deterioration of his condition will be evident later on.

Blood Examination.—On entrance his leukocyte count was 12,200, his hemoglobin 75 per cent. On the 22d a second leukocyte count was 12,800.

History.—The history which we have been able to obtain is very meager because the patient is unable to tell us anything about himself on account of his condition, and no friends have been sufficiently intimately associated with him just preceding the onset of his illness to give us any data. The patient has been living alone for some years, being separated from his wife and grown children, with whom he has not communicated for quite a long period of time. He is sixty-two years old. We have learned that his father died about the age of ninety of paralysis; his mother died of a cause which we have been unable to learn. He has a brother and sister living and well, and has five children, all of whom are living and well. There is no history of carcinoma or tuberculosis in the family.

Up to fifteen years ago the patient was in excellent physical condition. At that time he is said to have suffered from paralysis of one side of his body. A son who appeared at the hospital a day or two ago does not remember which side it was. The patient was then forty-seven years old. The paralysis gradually improved, but the gait was always slightly impaired. The son says that the treatment given him at that time consisted of electricity and baths. Four years ago he had a very severe attack of pain and was delirious. His son states that it was an attack of kidney trouble which came on suddenly and from which he recovered after he had been given some sweats. Last November he had an attack of bronchitis, went to Florida, and returned two months later much improved. That is all the son knows concerning his condition, until two weeks ago when from a state of com-

parative health he passed overnight to the state in which he now is. His present illness began at 4 A. M. on Tuesday, March 14th.

DR. MIX (to Patient): How are you feeling to day?

PATIENT: I believe the way you made those things to him, I just not over it was the one pair. (Takes doctor's glasses and puts them on nose.) It's too much, it's too much.

DR. MIX: In this act he shows no apraxia. He knows what those glasses are, he knows what they are for, and he puts them on in order to put them to the proper use. (To Patient—handing him a pencil): Can you write?

PATIENT (attempts to put the pencil on for glasses and then looks at history sheet): You got some more, I do not, you can see for yourself pretty fair.

DR. MIX: In this second act he does show apraxia. He is trying to put the pencil to a use for which only glasses are intended. (To Patient): Can you read this?

PATIENT: I can with everybody.

DR. MIX: He says "everybody" and "I believe" hundreds of times a day, saying them over and over, and jumbling them into sentences in all sorts of relations. A few days ago his physical condition was much better than it is at the present time and every few moments he would say in a pleasant sort of way as a Frenchman might say, "Mon Dieu," "Oh, my God! Oh, my God!" If you gave him a pencil he would smile and apparently try to write, but he was not able to write anything that was readable. The attempt to put the pencil upon his nose for a pair of eyeglasses is one of several evidences of apraxia which he has shown. By "apraxia" we mean an inability to recognize the use to which an object should be put. (Hands patient pencil.)

PATIENT: Nobody here but can talk. (Looks at history sheet again.) Of course, I am not exactly like anybody else, you understand, I am not.

DR. MIX: Please note how excellently he modulates his sentences. He evidently believes he is speaking straight to the point.

Now what ails him? He has a condition which is rather characteristic and it is possible to come to some clear conclusions from

the few data which we have thus far obtained. What happened to him suddenly at 4 o'clock on the morning of March 14th?

VISITING PHYSICIAN: He had a hemorrhage or an embolus.

DR. MIX: And a third thing which he might have had is a thrombus. If it were an embolus, where would it come from?

VISITOR: The heart.

DR. MIX: Do most *emboli* come from the heart? The answer to this question depends upon the age of the patient. If the patient is a young individual with a rheumatic endocarditis the probabilities are that the emboli come from the endocardium. Ulcerative endocarditis is almost sure to produce large numbers of emboli which frequently occur in showers. We also find emboli in cases of more or less chronic mitral stenosis with decompensation. A mitral stenosis with decompensation of any degree of intensity is also apt to lead to the formation of a clot in the heart itself. This may be swept out into the blood-vessels and plug one of them. Ordinarily, clots which start in this way pass up in the left common carotid artery and are lodged in the left side of the brain.

Sometimes thrombi form in the heart chambers after a period of myocardial weakness. You watch the patient and you struggle to improve his cardiac condition. You probably succeed. Just as you think he is starting on the road to recovery his heart will wring itself free from a large parietal clot and the patient will die within a few hours or a few days from cerebral embolism. I think I told you of a patient in whom such a clot formed in the heart. This patient had been ill for some weeks with intense dyspnea, cyanosis, and delirium cordis. Gradually we were able by rest and appropriate medication to straighten out his arythmia so that the heart was beating at a rate under 100 and with a fair degree of regularity. I left him one evening feeling particularly well. At 10 o'clock that night he suddenly became unconscious. The heart had freed itself from a large clot which passed up his right innominate artery. At the bifurcation of the innominate the clot evidently broke, part of it passing into the axillary and cutting off the right radial pulse, and the other portion of the clot passing into the right common carotid and cutting

off the supply of blood to the right side of the brain, thus causing a left-sided hemiplegia. This is the only instance that I have encountered of a very large clot leaving the heart and slitting into two main portions. Of course, this patient of whom I have just spoken lived but a few hours and never regained consciousness. Such large thrombi occur in cases of mitral regurgitation with intense decompensation, but they are more apt to occur in mitral stenosis. It is likely that these large clots form within the meshes of the chordæ tendineæ; then, as the heart begins to regain its strength and contraction overcomes the dilatation, the very reduction in size of the heart liberates the clot and sends it out into the blood-stream. Many times just when the patient seems to be on the road to recovery such a thrombus is hurled from the heart and becomes a deadly embolus.

We have, then, as **causes of emboli,** mitral disease, ulcerative and malignant endocarditis, and thrombi occurring in cases of myocarditis and decompensation in valvular disease. You might think that aneurysms would be a fruitful source of emboli, but they are not. Emboli from aneurysms are uncommon. I do not mean to say that they never occur, but I do mean to affirm that they are not so frequent as you might at first thought expect them to be.

Perhaps the most fruitful source of emboli is arteriosclerosis of the large blood-vessels. These emboli occur in people of advanced age who are suffering from arteriosclerosis. A very common location for the development of these thrombi is at the point where the internal carotid enters the cranial cavity from the cavernous sinus. You will remember that it immediately splits into four branches: the anterior choroid, communicating, the anterior, and middle cerebral arteries. At this point, therefore, it rapidly changes its diameter; and for some reason or other, probably because at points of bifurcation arteries are subjected to the greatest strain, the arteriosclerotic process begins at this point. If you have the opportunity of seeing postmortem examinations in individuals showing arteriosclerosis you will very frequently find a hard patch at the point where the internal carotid breaks up into its four terminal branches.

In arteriosclerosis of the aorta it occasionally happens that parietal thrombi form which break off and are carried into the blood-stream. Usually these make their way to the brain arteries and produce various types of cerebral lesions. Frequently, too, the thrombi develop just at the point of the arteriosclerotic process affecting the internal carotid. Occasionally one sees a large thrombus growing in the middle cerebral artery and cutting off a large part of the supply to the cortex of the brain. Frequently small emboli, breaking off from parietal thrombi, pass into the median ganglionic system and produce hemiplegias by affecting the internal capsule.

It is by no means unusual for thrombi to reach their maximum development between the hours of 12 and 4 A. M.—a time when the blood-pressure usually reaches its minimum. It is evident that at such hours it is easier for thrombi to form than during the daytime or during periods of stress. We consequently find in cases of pure thrombosis that the paralysis is discovered on waking. We also find that cases of thrombosis with secondary embolism develop the paralytic phenomena which they give rise to suddenly and abruptly, just as in cases of hemorrhage. An embolus is always dislodged suddenly, and it is forced into the artery, which it plugs with extreme suddenness. The result is that it is as rapid in its work as a hemorrhage. It also happens that it is apt to occur when the body is under physical strain. Emboli, therefore, are more apt to occur during the daytime than in the sleeping states.

On the other hand, thrombi are formed most readily when the blood-current is weakest, and are more prone to develop in the early morning hours. They do not produce symptoms in a moment, but they give rise to their symptomatology somewhat gradually. Frequently a paresthesia of the arm indicates an oncoming thrombus. This is particularly true in younger people suffering with cerebral syphilis. Thrombus of a blood-vessel first produces an exceedingly mild cortical ischemia with paresthetic sensations in the extremities. These paresthetic sensations are gradually replaced by motor irritative phenomena, and then finally there comes the complete motor paralysis, as the

cortex is more and more deprived of blood by reason of the thrombus in the artery of supply.

Usually a patient who suffers from thrombosis does not know he is paralyzed when he wakes up in the morning until he makes the effort to get out of bed. Occasionally such an effort will dislodge a thrombus and sweep it further along in the artery and then suddenly plug it. Thus, the patient may rather suddenly be attacked with a stroke just as he gets out of bed in the morning. Thromboses usually produce paralyses which are monoplegic in type because, as a rule, they are cortical.

In this patient there is no paralysis of the arms and legs, but there is a very marked disturbance of speech. This particular variety of disturbance is known as *paraphasia*, and it is due to a particular lesion which was first described by Wernicke, and which sometimes goes by the name of *Wernicke's conduction paraphasia*. It is possible to make by the character of the talk this man has given us a fairly definite diagnosis as far as localization is concerned.

I think from what I have told you that we may assume that this individual, sixty-two years old, went to bed in his usual health Monday night, March 13th. At 4 o'clock in the morning of March 14th a thrombus had formed in such a position as to give rise to the group of symptoms this patient presents. He woke up as usual, but he woke up apparently deprived of his senses. He was not paralyzed, he had the full use of his arms and of his legs, but he had a marked speech disturbance. He was suffering from aphasia.

Varieties of Aphasia.—Of aphasia there are many varieties. This patient does not show motor aphasia because he is able to speak. If he was unable to utter a word we would say that he was suffering from *motor aphasia*, or *aphemia*. Possibly in his case there is also *auditory aphasia*, because when I tell him to put his tongue out he does not do it.

Dr. Mix (to Patient): Put out your tongue. (Patient fails to do so.)

Patient: How can I remember exactly? I have one and three-quarters, but that is all. I do not believe that was good, but not to anybody else.

Dr. Mix: It is evident that there is auditory aphasia in his case. He evidently hears, but he does not know what the words mean, and he begins to talk because I spoke to him. He has the motor capacity of putting out his tongue, but he fails to understand the directions which are given him to do so.

Speech is not only spoken and heard, but it is also written. Another variety of aphasia is known as *visual aphasia* or *word-blindness*. In this condition individuals are unable to read things that are written. If we ask this man to read something for us

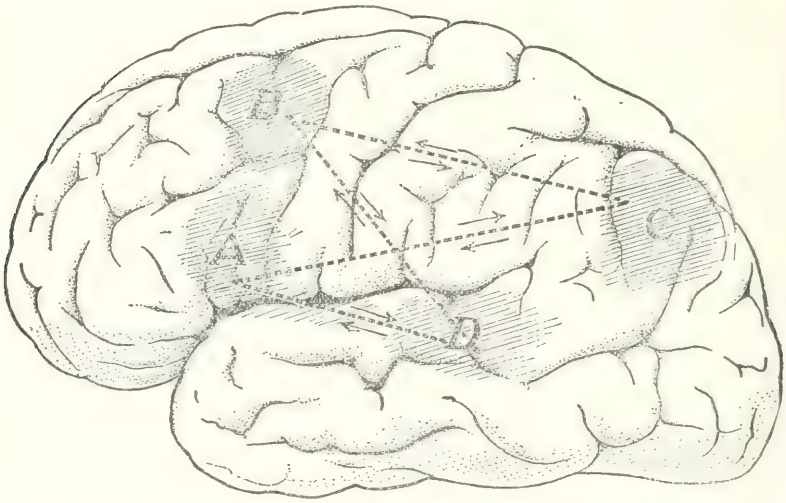

Fig. 8.—*A*, Motor speech center; *B*, Exner's writing center; *C*, word-seeing center; *D*, word-hearing center. Dotted lines indicate association pathways. Arrows indicate directions of conductivity.

he cannot even tell what the letters are. He has, therefore, no motor aphasia, but he does have both auditory aphasia or word-deafness, and visual aphasia or word-blindness. This is a fairly frequent combination clinically.

Cerebral Localization.—We may get a better idea of the disturbance which this man is suffering from if we have upon the blackboard a diagram giving us the various cerebral locations. Please note the locations for the word-speaking center, Exner's word-writing center, the center for the reading of words in the

gyrus angularis and supramarginalis, and the center for hearing situated in the temporal lobe. The existence and location of Exner's writing center has been disputed. It is usually located in the posterior portion of the second frontal convolution in juxtaposition with the ascending frontal. If we grant the existence of Exner's writing center, there are, then, four main centers that have to do with words: one with word hearing, one with word seeing, one with the reading of words out loud, and one with the writing of words.

Development of Association Pathways.—Between these four main centers there are certain pathways of association. When you go to school these pathways are developed for you by the teacher. One of the usual means of instruction in the schoolrooms is to have the child repeat what the teacher says. The words are heard by the child in the word-hearing center and they are repeated in the motor-speech center. The teacher says, "Now say after me what I say," and the scholar hears it in the word-hearing center and speaks it in the word-speaking center, and thus establishes a pathway between the word-hearing center and the word-speaking center. When the child speaks the word, the child also hears the word that it speaks and knows whether it has made a mistake or spoken the word correctly. In other words, the association pathway is simultaneously established from the word-speaking center back to the word-hearing center.

Another method of instruction is to have the child read from a book. He reads the words by means of his word-seeing center. He pronounces the words that he reads by his word-speaking center. He thus develops the association pathway between his word-seeing and his word-speaking center. Furthermore, as you will discover by stopping to think a moment, a similar association pathway runs in a reverse direction between these two centers. We speak a word and we can visualize it in our word-seeing center. Sometimes we misspell a word orally, then write it, and recognize our error and correct it.

Another method which teachers are fond of making use of in instructing children is writing from dictation. The child hears the word in the word-hearing center and puts it on paper in Ex-

ner's word-writing center. In other words, the child writes by means of the word-writing center that which the child hears in the word-hearing center when the teacher dictates. Children also copy from copy-books. In this case an association pathway is formed between the word-seeing center and the word-writing center. The child sees the word in the copy-book and by means of the word-writing center puts the word upon paper.

These association pathways have been known from almost time immemorial to teachers, and they are made use of in all systems of education. In Arabia the children learn the Koran by reciting it after the teacher. In our higher systems of education students learn to write French or German from dictation. They learn to write French and German by means of the association pathways between the word-seeing and the word-writing centers just as in their very early life in the kindergarten or primary school they get their first rudimentary knowledge of the English language.

There are other pathways which are not so well trained as these fundamental ones. For example, we can see a thing in a book and hear somebody reading it and recognize whether it is read correctly or incorrectly. In other words, we are really making use of the seeing center and the hearing center; thus an association pathway is established between these two centers. Teachers develop this pathway. The teacher sees the words in the book, the child recites those words, the teacher hears them, and develops the association pathway.

As a rule, these association pathways connect up sensory centers with motor centers or *vice versa*. Motor centers are not connected with each other directly and neither are sensory centers. For example, a motor center such as the speech center is connected either with the word-hearing or the word-seeing center, and, as a rule, there are poorly developed association pathways between the word-writing and the word-speaking center, and between the word-seeing and the word-hearing center.

These word centers and association pathways are developed only on the left side of the brain in right-handed people. The explanation which is given for this state of affairs, and which I

believe to be correct, is that in fetal life the very best blood-supply goes straight up through the left common carotid artery to the left side of the brain. You will remember that when the blood passes from the left ventricle to the aorta the first branch which it reaches is the innominate artery. The total blood passing into the innominate artery is divided between the right side of the brain and the right subclavian artery. On the other hand, the blood which goes to the left side of the brain passes up a straight trunk line through the left common carotid artery. As a matter of fact, it is thought that the total amount of blood reaching the left side of the brain through the undivided left common carotid artery is greater than the blood-supply which reaches the right side of the brain through the right innominate artery and its branch, the left common carotid. Thus it happens that the left side of the brain gets a little better and a little earlier start, and it is on this account that the speech centers are developed in the left side of the brain. If a person is left handed, it usually indicates that in fetal life the right side of the brain got the best and earliest blood-supply. Hence it is that in left-handed people the speech centers develop in the right side of the brain. In other words, the side of the brain which develops the speech centers is the side which is best supplied with nutrition in early fetal life. This patient is right handed, and the lesion, therefore, is on the left side of the brain.

His writing center is probably not involved. He does not write, but I do not know absolutely whether it is because he cannot write or because he cannot get the words through from his sensory centers. He can use a lead pencil reasonably well, but he cannot make letters that anybody can read. Probably the motor centers in his case are uninvolved, but he cannot make use of them because the association pathways to them are injured.

Now what happened to him? Evidently at 4 A. M. Tuesday, March 14th, a thrombus developed somewhere in the left side of his brain which interfered with his word-seeing and word-hearing centers. Besides interfering with these centers it also interfered with the association pathways leading from them. The location of the association pathway between the word-hearing center and

the word-speaking center has been located by Wernicke as lying beneath the island of Reil in the region of the external capsule. When this association pathway is destroyed the patient talks nonsense. The earliest of all the association pathways developed by pedagogy is the one by which the child repeats what is said to him. In other words, he makes use of Wernicke's speech tract.

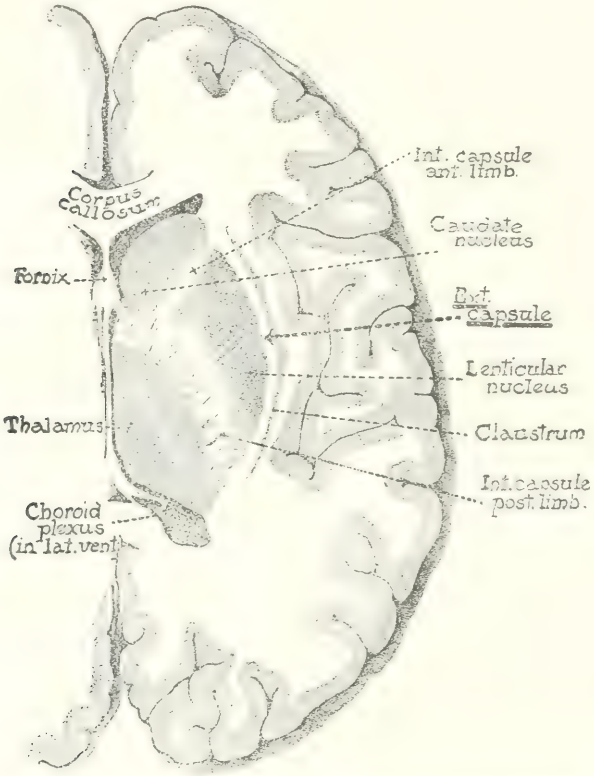

Fig. 9.—Wernicke's association pathway passes through the external capsule.

This man is unable to make use of that tract. He keeps right on talking and evidently seems to himself to be saying sentences, but there is no sense in them. This is because he has lost the ability to correlate between his word-hearing center and his word-speaking center. When you hear yourself talking you know what you are saying and you know whether you are saying it correctly.

Occasionally we misspeak ourselves because there is a slip-up in our association pathway between our speaking center and our hearing center, but we ordinarily correct ourselves because we hear ourselves making the mistake. For example, I hear all that I am saying, and if I make a misstatement I correct myself, but this man cannot recognize the words he is saying, and while he thinks he is talking sense he is talking nonsense. It is because he has a disturbance in the pathway between his word-hearing center and his speech center which association pathway lies in the *external capsule.*

Suppose this (illustrating on blackboard) is the midline of the brain: Here lies the caudate nucleus, here is the optic thalamus, there is the lenticular nucleus, and between the lenticular nucleus and the caudate nucleus and optic thalamus is the internal capsule. Just outside of the lenticular nucleus is the external capsule covered by the claustrum, outside of which again lies a slight layer of medullary cortex and then the cortex of the island of Reil. The pathway which connects the word-speaking and the word-hearing center lies in the external capsule, and that area has been involved in this case. There is also disturbance in the temporal lobe in which lies the word-hearing center, and in the gyrus angularis in which lies the word-seeing center, for he has both word-blindness and word-deafness.

Conclusions.—At this time it is evident that we can conclude that the patient's case developed somewhat in the following manner: Monday evening, two weeks ago yesterday, this man went to bed apparently in perfect health. At 4 o'clock Tuesday morning he was suddenly seized with an inability to talk coherently. He was thought to be delirious or out of his head. He was brought to the hospital and it was at first difficult to say whether he was suffering from a brain condition or from uremia. He was not unconscious, as one would expect him to be if he were suffering from a cerebral hemorrhage. The fact that he was not unconscious made it more likely that he was suffering from a thrombus than from an embolus or hemorrhage. Physical examination showed no disturbance except the old paralysis which he had fifteen years ago. Examination of the urine showed that the

diagnosis of uremia was untenable. Further consideration of his case led to the discovery of the apraxia and of the aphasia, which upon analysis was found to be due to word-deafness and word-blindness with the involvement of the association pathways between these centers and the word-speaking and word-writing centers. Thus we were enabled to conclude that a thrombus had produced Wernicke's combined aphasia.

Moreover, the patient confirmed our idea of thrombosis three days ago by developing another very serious thrombus and embolus. This took place in the popliteal artery where this artery gives off the anterior tibial branch. Such a *popliteal thrombus* is usually manifested by a good deal of pain, by discoloration, and by coldness. I want you to look at his leg. It is going to take his life, probably within a few days' time, because operation has been refused by his friends, and I think with reason, considering his brain condition. The family very decidedly objects to having the leg amputated. You can see the difference between the two legs at a glance. You can notice how blanched the toe is on the affected foot, and if you feel it you will notice that it is very cold. Within a few hours it will become gangrenous. There is no pulsation in the posterior tibial where it passes around the internal malleolus, and there is no pulsation in the dorsalis pedis artery.

Where did this embolus come from? Presumably from an arteriosclerotic process affecting the aorta. Probably the same focus which gave rise to the thrombus which passed up into the left common carotid artery gave rise to this thrombus which lodged in the popliteal artery. In all cases of thrombus which lead to coldness and loss of pulsation in the posterior and anterior tibial arteries we know immediately that the trouble is at the point where the popliteal gives off the anterior tibial artery. It is at this point that the embolus almost invariably lodges. Surgeons have, therefore, learned that in such cases there is no use in amputating a toe, and then subsequently amputating at the ankle, and then possibly half the leg. They know that it is necessary at the very first to amputate at the end of the femur. The shock of such an amputation is tremendous, and many

patients do not recover from it. In this case, in which emboli have formed within twelve days' time, one passing to the brain and the other to the left leg, it does not seem wise to operate.

DR. MIX (to Patient): Tell us about your leg.

PATIENT: I say if anybody knows this I am able to have the same they have done, but can't get their kind, I have—

DR. MIX: What do you think about it?

PATIENT: It's blond, yes.

DR. MIX: You can all see how stained and mottled the leg is. There is no circulation in it, and shock, sepsis, and intoxication will follow. The temperature does not rise very high in these cases, but the intoxication is tremendous and usually carries the patient off. Many of these patients develop a hypostatic pneumonia and are carried off in that manner. In the case of this patient it is doubtless a blessing to him that he had the cerebral upset first, because by anesthetizing his brain it has kept him from the realization of the great pain which he otherwise would have felt because of the thrombus in his leg. Usually a great deal of pain is complained of by these patients, but the other day this man was slapping his leg and saying, "Nothing, nothing, nothing." I knew he meant that the leg was without sensation, but the vigor with which he slapped it seemed to indicate that he had very little feeling in it.

The patient told us about this leg in his own way four days ago, and twenty-four hours later even the nurse noticed it and told me that there was something the matter with his leg.

Treatment.—Nothing can be done medically for such a case. Stimulants, such as strychnin or digitalis, are useless because blood cannot be driven through the popliteal artery. I have dissected the popliteal artery in the case of one patient and found a cord of clotted blood several inches long running from the popliteal artery halfway down through the posterior tibial. This patient lived three or four days and then died from the shock of the operation. The mortality from operation in this condition is high, and in view of the fact that this patient has disturbances of a similar thrombotic nature in his brain, it is evidently unwise to

advise operation. We have placed the facts before his son and the latter has made the decision for him.

PATIENT: I know that this thing if I could a difference, anybody could have, but I haven't.

DR. MIX (to Patient): What do you think about an operation?

PATIENT: They can do it just as good, see? If you have not got it you can find somebody.

DR. MIX: All through his talk he seems to be trying to express some idea. I think he himself is in favor of an operation, and he thinks that if we do not do it we can get somebody else to perform it for us. (To Patient): Do you think we had better cut the leg off?

PATIENT: If you have not got anybody.

DR. MIX: What do you think we had better do about it?

PATIENT: Anybody can help, see? Sure.

DR. MIX: If the patient could only talk properly we might get along splendidly, but since he cannot talk intelligibly we shall have to do the best we can without his assistance. If we could make him understand, put the question to him and let him decide it, it would be all right, but we have no legal right to say that he shall be operated upon. That right rests with the patient, and inasmuch as he is not able to decide it, the family must take the risk of doing so for him.

CLINIC OF DR. ISAAC A. ABT

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FEEDING THE NORMAL BABY. BREAST FEEDING

Definition of Turgor and Tonus as Applied to Infants. Breast Milk the Natural Food for the Baby. Significance of Colostrum—Technic of Breast Feeding. Maternal Conditions which Contra-indicate Nursing. Causes of Failure in Breast Feeding. Unreliability of Testing the Chemical Composition of Breast Milk. Effects of Overfeeding and Underfeeding at the Breast. Effects of Constitutional Anomalies on Nutrition of Infants. Advantages of Breast Milk for Sick Infants—Detoxication—Reparation Period. Use of Laxatives in Newborn Baby is Irrational.

November 6, 1915.

LET us devote a portion of the hour to a consideration of the *normal infant*. You note the happy and contented appearance of the baby. A healthy infant appears happy. As a rule, contentment in the infant is an indication of good health. A baby is by nature optimistic; all the world looks beautiful to him.

This baby is seven weeks old and is nursed by the mother, who is in the surgical ward. The temperature line never goes above 99° F., and for the most part is between 98°, 98.2°, and 99° F.; in other words, a line which we would say represents a condition of *monothermia*. Now, besides that, this baby has a good color; it weighs 11 pounds 11 ounces, and we notice that the baby is well covered with subcutaneous fat. On feeling the baby's body one has the sensation of a certain tenseness, a fulness, and this condition we call *turgor*. Now what is turgor?

Turgor is a term used in physiology and botany. In physiology it refers to the normal fulness of the capillaries and smaller

vessels, upon which is supposed to depend in part the resilience of the tissues; it is usually qualified by the epithet "vital." The state of turgor is a condition referred to by botanic physiologists, by virtue of which the framework of the protoplasm of the plant retains its content tenaciously. Loss of turgor in an infant means disease, and occurs when there are nutritional disturbances or when there is wasting.

We speak of *tonus*. Upon feeling a baby's muscles, for instance, the abdominal muscles, we feel there is some resistance, some tenseness. This tonus is a specific attribute of muscle. Tonus refers to the tenseness or tension of resisting muscles.

Nutritional disturbances may cause changes in living muscles. For instance, the muscle may be rigid or atonic, dependent upon alimentary disturbances. A muscle may be hypertonic if a baby is fed for a long time on starch. We speak of this as one of the manifestations of starch injury. The baby becomes rigid on account of muscular tonicity. On the other hand, in some nutritional disturbances the muscles may become atonic.

So we judge the health of the baby by monothermia, by its general condition, by the psychic condition—if the baby is happy or fretful—by the normal turgor of the body, by the tonus of the muscles, and then, too, by the respiration and by the heart's action. A normal healthy baby has well-defined, clear-cut tones; the baby that is suffering from marasmus or malnutrition may have some variation in the first heart tone, the tone may be weak or, in severer cases, may be almost inaudible.

Breast Feeding.—Now this morning I want to point out to you some points connected with breast feeding. It is an important matter and deserves your most careful consideration.

Shortly after birth the breasts secrete a yellow, cloudy, sticky secretion which we call *colostrum*. The yellow color is characteristic of this early period and does not appear later, not even in the period of ablactation. The color is due to a yellow pigment substance which combines with the fat. The exact nature of the yellow pigment substance is not known. The microscopic examination of the colostrum shows a variety of fat corpuscles and irregularly formed clumps of fat drops of various sizes, and

also some mulberry cells containing fat, the so-called colostrum corpuscles. Most authors believe with Czerny that the colostrum cells are derived from leukocytes and that they have the function of extracting fat out of the mammary gland.

Significance of Colostrum.—If we stop to examine the difference between colostrum and mature breast milk the following points are most important:

(*a*) Colostrum is more concentrated and more viscid. It contains more protein, particularly more coagulable albumin.

(*b*) It is poorer in milk-sugar.

(*c*) The fat shows less perceptible quantitative differences, though it is distinguished from that of mature milk in that it contains a greater quantity of oleic acid, and in that it combines with a yellow dye-stuff.

(*d*) Colostrum is relatively rich in lipoids.

(*e*) The mineral salts are more abundant. Colostrum is relatively poor in the earthy alkalies.

(*f*) The ferment content, as well as the quantity of immune substances, is increased.

Now, if we ask whether colostrum has an especial significance for the newborn infant, or if the infant is benefited by first receiving colostrum, then transitional milk, and finally pure breast milk, we find the question difficult to answer, though on general principles one would be inclined to believe that the natural arrangement was necessarily the best adapted for the physiologic needs of the infant. It has been pointed out that colostrum, a concentrated food containing large quantities of protein and fat, would supply in the most exquisite way that great heat loss which is sustained by the newborn infant, and would provide for the necessary heat production.

Birk has called attention to the fact that the proteins of colostrum milk show a close relationship to the serum albumin in biologic aspects, and that the feeding with colostrum protein might in a sense be considered a continuation of the food supply to the infant by which nutrition is carried by the blood through the umbilical cord. In brief, it may be said that *the advantage of colostrum milk for the young infant consists in the*

fact that he receives in a small quantity of food a relatively high caloric value in nutritional substances. Colostrum is said to be mildly laxative to the infant. It is not probable, however, that the colostrum produces the intestinal irritation which is so commonly ascribed to it, and altogether it has not been shown either clinically or experimentally that the colostrum milk produces laxative effects.

The next point refers to **breast milk** and **sucking of the infant:** The breast secretion may be established in two ways. In one group of cases the glands fill gradually, the breasts become larger, the mother experiences a feeling of fulness, and milk may be expressed either in streams or in drops. The first day the infant receives very little, and on the third or the fourth day the flow has been established. By this time the milk has lost its colostrum character and begins to look like a white milk.

In the second type the secretion is established suddenly with marked symptoms. The milk, so to say, shoots in; the breasts become hard, increase in volume; the mother complains of a feeling of fulness and pain. Moderate pressure causes pain. This occurs on the third or fourth day, occasionally as early as the second day.

Between these two types many variations may occur. As a rule, secretion is established by the fourth day.

Some authors speak of two varieties of breasts: (*a*) Those in which the milk flows easily; (*b*) and those in which it flows with difficulty. The ease or difficulty with which the breast may be emptied does not always depend on the quantity of milk secreted, for a feeble baby may have difficulty in nursing, though the breast is secreting abundant milk. A slow breast does not always depend upon the infant, but may depend upon certain circumstances which are acting in the mother. A nervous woman may inhibit the flow of milk, and many authors think that in these cases when lactagogues are said to be successful they act as suggestive agents by instilling courage and hope into mothers.

Technic of Breast Feeding.—I am not going into any detailed account of the technic of breast feeding, but will give you the

important points which represent the consensus of opinion on this subject.

1. The young, inexperienced mother should be instructed in the most elementary details concerning nursing. She is shown how to retract the parenchyma of the breast from the nipple so that the infant's nose will not be buried in the mamma and respiration will not be obstructed in this manner. If she trains the baby to grasp the areola as well as the nipple, the milk flows more freely and the nipple is less liable to be traumatized and rendered painful. The nipples should be kept scrupulously clean and may be washed before nursing, using plain water, at least not strong antiseptics.

2. Washing the baby's mouth is obsolete and has been generally abandoned.

3. As a rule, the infant should be fed from only one breast at a time. Only in those cases where the milk is insufficient in quantity should both breasts be given at one time.

4. Long feeding intervals—four hours—are advised for normal babies, five or six feedings daily, though individual variation may be necessary in cases where the infant is ill or where the breast milk is insufficient.

5. The quantity of breast milk received by the baby at a single meal varies within wide limits. The largest quantity ingested at one meal may be two or three times as great as the quantity taken at another feeding on the same day.

6. The food of the mother during lactation should be sufficient, but not excessive. She should drink fluids in proportion to her thirst. The excessive drinking of milk and beer, as is so frequently recommended, is unnecessary, though if a woman is accustomed to partake of beer in moderation no harm will come to the baby or its food-supply.

7. It is equally important to avoid underfeeding the nursing mother. Czerney and Kellar believe it an error to materially reduce the diet of a puerperal woman. They believe that a reduction of food and water may lead to lowered milk secretion.

8. Nearly every writer on subjects related to maternal nursing has declared against the view that certain foods ingested by the

mother are capable of producing disturbances in the infant through the breast milk. Raw food, fruits, and gas-producing vegetables may be eaten by the mother if they do not cause discomfort to her. They produce no changes in the composition of the breast milk.

9. It is quite indifferent whether or not a baby is put to the breast during the first or second twenty-four hours. The amount of milk in the breasts during this early period is, as a rule, so small that it need scarcely be considered from a nutritional point.

10. Drugs may be transmitted to the baby by breast milk secretion, though the quantity eliminated in any case must be infinitesimally small.

Immunity and Breast Feeding.—It may be said that breast feeding confers a certain antitoxic protection which is conveyed from mother to child. It has not been proved up to the present time that the immunity is due to bacteriolytic processes. It is possible that the food acts indirectly upon the cells which produce antibodies, and in this way a specific immunity is brought about. According to the investigations of Moll, we know that the young animal organism has much less power to produce antibodies than the adult, and therefore the resistance against infection and disease processes is relatively diminished in the infant.

Nutritional Disturbances of Breast-fed Infants.—There is a widespread feeling among the laity and physicians that if an infant does not prosper at the breast, if he does not show a constant increase in weight, or if the infant's stools are green or thin or frequent, that there is a basic derangement in the composition of the breast milk or that it contains toxic substances, and the baby is forthwith weaned.

The chemical examination of breast milk which has been extensively resorted to by clinicians in order to determine whether the milk was of the proper chemical composition, and also if it was suitable for the infant, has fallen into disuse. Clinicians themselves know that milk which gives an average or normal result in analysis may disagree with the baby, while milk which responds, so to speak, unfavorably to chemical tests with

low fats or high proteins or fats, frequently agrees better than the milk with a more ideal or normal chemical analysis.

This test has proved to be altogether unreliable. The chemical composition of milk varies from hour to hour and also during the milking period the component substances vary. The first milk is different from the middle milk, and the middle milk differs from the last portion.

Nevertheless, every practitioner knows that babies do poorly at the breast at times. There is a class of mothers who secrete little or no milk. These we will speak of later as cases of agalactia. Mothers who secrete insufficient milk we will again refer to as cases of hypogalactia. Undoubtedly there are occasionally women who secrete milk which is chemically or biologically defective. This is shown clinically by vomiting, diarrhea, or other signs of illness in the baby. The prevailing view is that this condition is rare. We cannot definitely classify or recognize such cases from any known method of milk examination, chemical or biologic.

We may at this point refer to some experimental work on the nature of the *fat content of milk*.

Moll thought that by feeding bacon to mothers the fat content of the milk was increased. Engel, commenting on the work of Moll and speaking from the result of experimental work, says that the results of Moll's observation are incorrect. He thinks that the fat content of breast milk cannot be increased by alimentary influences. Engel has found that the largest quantity of breast milk is secreted in the morning, but that a considerable quantity is secreted in the evening. These milks are low in fat content. Between these two periods the milk is less in quantity, though fat is high. These tests are derived from wet nurses in institutions who are nursing several infants.

Engel concludes that a superfluous amount of fat fed to the mother is of no use and tends to produce corpulence in the mother. Since the morning milk and the evening milk are greater in quantity than at other times, and since the fat content is especially low at these periods, such milk should be chosen in institutions where wet nurses are kept for therapeutic purposes.

It is claimed that infants who receive a milk which is constantly high in fat, gain more slowly than infants who receive a milk with a lower fat content.

Maternal Conditions which Contra-indicate Nursing.—It is generally agreed by all authorities that an open tuberculosis in the mother is a positive contra-indication to nursing. But, on the other side, mothers who are suffering from acute or chronic infections or constitutional disorders may secrete normal germ-free milk which the baby may take with impunity. Such milk may be given to the baby by bottle. Finkelstein suggests that in erysipelas of the mother the breast milk may be boiled. As a matter of fact, the problem of continuing a baby at the breast of a woman with an acute infectious process depends upon the strength of the mother and the contagiousness of the illness.

In considering lymphangitis and mastitis some authorities think that the affected breast may be emptied by the infant or the breast-pump if the milk is free from pus. Obstetricians, as a rule, put the breast completely at rest, suspending its functional activity.

If temperature disappears in the mild cases in three or four days breast feeding may be resumed.

Causes of Failure in Breast Feeding.—We not infrequently observe an infant who fails to prosper at the breast, though milk is being secreted in abundance. Some causes of failure in breast feeding may be explained by the occurrence of mild infections in infants, the so-called *parenteral infections*. It is a matter of general observation that a pharyngitis, furunculosis, otitis, cystitis, or a gastro-intestinal infection will result in failure to develop, as well as disturbances of digestion in breast-fed infants.

Mild dyspeptic attacks of short duration may be caused by slight external causes—insufficient warmth or excessive heat—overfeeding or underfeeding.

To what extent the quantity of the milk may be altered by emotion or grief, by fatigue or menstruation so as to cause temporary digestive disturbances in the baby is still under discussion. The foremost pediatric teachers declare that milk is not per-

manently harmed by the conditions referred to. The baby does not fall ill because the mother is menstruating or is nervous.

Bendix says that he would accept with much skepticism any statements which refer to permanent deterioration of milk because of menstruation. He found no change in the breast milk, though occasionally there was a diminution in quantity, and he frequently found an increase in the fat content. He concedes, however, that during the period, or a portion of it, the baby may be restless or refuse the food, or show signs of mild dyspepsia with vomiting and diarrhea. Statistics show that 50 per cent. of nursing women are amenorrheic, 43 per cent. menstruate, and of these 20 per cent. have regular periods during lactation. In a word, then, many women menstruate during lactation without producing any effect on the baby. Occasionally a nursing infant suffers from mild dyspepsia during this period.

Every practitioner, I am sure, has observed temporary disturbances in the breast-fed baby where the mother has been subjected to some violent grief, some overpowering emotion, a most fatiguing social function, or a night spent in dancing, drinking, and dissipation. The effects on the baby are, as a rule, mild and of short duration. They consist of vomiting, diarrhea, restlessness, colic, and possibly a slight fever reaction.

Overfeeding at the Breast.—Overfeeding is a frequent cause of alimentary disturbance in nurslings, and may result in injury to the organism.

Nearly every mother primarily interprets the cry of her infant as due to hunger. If the mother yields to her feelings and feeds the baby nearly every time it cries, a condition of overfeeding readily occurs. Or in those cases where the milk secretion is overabundant and the infant is very vigorous, overfeeding may occur before it is noticed. For a time at least every breast-fed infant tolerates an excessive quantity of breast milk. In a brief period, however, symptoms of food injury present themselves. The infants become restless, have disturbed sleep at night, and vomit occasionally. They remain stationary or lose in weight. They may soon become flatulent and show an increase in the number of stools, which are dyspeptic in character.

The diarrhea leads the mother to consult a physician. The stools when evacuated are green or become green on standing. They are not homogeneous, they are inclined to be lumpy, and contain mucous and soapy curds. Vomiting may be a most prominent symptom. The vomiting may consist of single severe attacks or it may be characterized by frequent spitting of a small quantity of food until eventually the weight disturbances occur; the infant loses his healthy pink color and his happy demeanor. Severe colicky attacks are not uncommon. Disturbances of metabolism may be noticed or irregularity of temperature and lactosuria and mild symptoms of intoxication may occur. These children desire to sleep, they show forms of circulatory weakness, or, indeed, actual alimentary intoxication may occur. In these cases, too, some parenteral infection may occur and the baby may be rendered seriously, if not fatally, ill.

The *treatment* consists, in the milder cases, of reducing the number of meals to five in the twenty-four hours, and limiting the time of the baby at the breast. In the severer cases the treatment consists of a short period of starvation and weak tea with saccharin or water, followed by moderate quantities of breast milk.

Underfeeding.—Another point, that is just the reverse or opposite of what I have just spoken, is underfeeding. The extensive literature and research on the disturbances of breast feeding have been for the most part devoted to the subject of overfeeding during the past few years.

We know, however, that it is possible that the error may occur in the opposite direction and that the infant may suffer just as severely from underfeeding. We know, too, that the withdrawal of food-stuff from the infant may result in more profound effects than in the adult. The baby may suffer from hunger which we obviously understand as a constitutional state due to the withdrawal of food. Underfeeding is due to the partial withdrawal of food, and this may be quantitative, in that the infant receives all the necessary compounds of food, but insufficient in quantity, or it may be qualitative in that the food does not contain the necessary component parts to maintain equilibrium and provide for growth.

Underfeeding may be due to a condition of *agalactia;* that is, a condition where the mother is supposed not to secrete milk. This condition of absolute inability to nurse is said to be extremely rare or not to occur at all. The condition of *hypogalactia*, where the mother secretes too little milk, does occur, and, according to some authorities, is found in 13 per cent. of all cases.

Or the milk flow may have been wholly established and after a time tends to disappear. We distinguish, too, a slow breast and an easy flowing breast. By the latter we understand a breast in which the slightest pressure causes relaxation of the sphincteric action of the milk-ducts, and a slow breast is one in which the sphincters remain more or less tightly closed, preventing the easy flow of milk.

The amount of energy which an infant must expend in emptying a slow breast results in general fatigue and he is likely to fall asleep at the breast. Very often this is interpreted to mean that the baby has had sufficient milk, and consequently in a short time he shows loss in weight. In other cases the milk appears late in the breast. It is the rule to assume that if the milk has not appeared until the fourth or fifth day of the confinement an agalactia has occurred, and no attempts are made to feed the baby at the breast, but it is known that milk may appear as late as the eighth day. Breast feeding should not be abandoned until every effort has been made to stimulate the flow of milk. This should be persisted in even as late as the twelfth or fourteenth day.

Hunger of Normal Breast-fed Infants.—It is known that infants may live a number of days without food. This occurs in congenital stenosis of the esophagus. In pyloric stenosis the infant may be practically without food for days or weeks. It has been shown by observation on animals that if they are allowed to starve, death will occur when they have lost two-fifths of their body weight. So far as infants are concerned, it has been noticed that if they lose 30 per cent. of their weight death is almost inevitable, though this would depend in part on the previous state of health of the infant.

A previously normal baby would offer greater resistance and

succumb less rapidly than a sick baby. An ordinary normal baby may be starved a day with scarcely any bad effect. On the second or third day of starvation the weight curve begins to fall, though the effects of hunger have a less marked influence on the weight curve of a normal baby.

It may be assumed that normal breast milk contains all the necessary ingredients to make the baby prosper. This is true until the eighth or ninth month of life. After this time it becomes a question whether the nutritive quality of breast milk is sufficient for a normal baby. We know that infants who are nursed exclusively after the ninth month are likely to develop anemia, and analyses have shown that breast milk is deficient in iron and calcium, and it has been thought that rickets under certain instances may occur in breast-fed infants because of the calcium deficiency. Czerny has suggested that the newborn organism comes into the world with a calcium depot and an iron depot, and in this way the deficiency in these mineral substances is supplied when the occasion demands. This thought is based on exact chemical analyses, though the hypothesis concerning the use to which the depots of iron and calcium are put requires investigation.

In a word, we may say that *the energy requirement of the infant is 100 calories per kilo, or 45 calories per pound.* If the quantity of food provides for considerably less calories than is indicated above, the foundation is laid for underfeeding.

When a baby is underfed at the breast the baby loses in weight and the regular weight curve ceases, depending upon the degree of underfeeding. The weight curve becomes horizontal or falls gradually. The abdomen sinks in and the muscles become relaxed. The skin becomes pale and lies in folds. Patches of eczema may occur. The temperature may remain normal or in some cases it may become subnormal. The heart's action may become slow, thus showing the effects of the underfeeding, and a slow pulse is a frequent clinical observation. The psychic state and the appetite are variable. In older infants who are underfed we note great unrest. They cry almost constantly and indicate a desire for food. Infants who put their fingers in their mouths

and make sucking movements, and show a facial expression of desire, and move their heads forward and backward as though they wanted something, we may safely assume are hungry. On the other hand, some underfed infants fall into an apathetic state. Such children lose in weight and have no appetite. Ordinarily, the underfed baby is constipated; occasionally he vomits. It is a fact that cannot be too strongly emphasized, however, that underfed breast babies may suffer from diarrhea, and that not infrequently such babies may do better if they are given sufficient food instead of too little.

We will now discuss underfeeding during the first fourteen days of life. I may say in explanation that the baby loses in weight after birth, and regains its birth-weight sometimes at the tenth day and sometimes at the end of two weeks and sometimes later, but in these cases where the baby is underfed at birth or shortly after birth, the baby fails to regain its initial weight. When you see many of these newborn babies you will make the observation that it is difficult to start them on feeding. It is difficult, first of all, because the physiologic function is sometimes interfered with by the great nervousness of the mother, or sometimes because the baby does not get the proper stimulus. It is an elemental principle in physics that the work done by the machine is equal to the work done on the machine. It seems to me that is what I learned a good many years ago. So it is with the breast—it may be regarded as a machine. The stimulation applied is of importance.

Some of these newborn babies will have considerable fever, and sometimes on the second, third, or fourth day the baby's fever is very high. It was Holt who first suggested that the febrile reaction was an *inanition fever*. We know, however, that during this early period of life there is a great loss of water from the body, and the fever is often abruptly terminated when the infant receives an abundance of water.

The underfeeding of breast-fed infants is largely influenced by the water intake. Those who receive sufficient water are less likely to feel the effects of the underfeeding as markedly as those who are receiving insufficient water.

The point is, give the babies an abundance of water to drink. There is one thing that may cause an error in diagnosis, and that is what?

VISITOR: Septic infections.

DR. ABT: Yes. *Septic infections* are sometimes very obscure, and, indeed, when the umbilical wound seems to have healed, a septic infection may be going on in the pharynx, ear, or where not, so that an inanition fever may be confused with a septic fever.

How do we diagnose underfeeding?

In the first place, it is possible to know how much a baby is getting. We note, for example, how the baby nurses; if it nurses vigorously or comes to the breast and shakes its head, or leaves it in a moment. Then, too, the mother feels how rapidly the milk is flowing from the breast; then we may weigh the baby a day before nursing and after nursing, or immediately before nursing and immediately after nursing, and the difference represents the amount of food ingested.

Moll has suggested that the urine of underfed infants at the breast be examined for the phosphorus. He has shown in a large number of cases that during health only a minimal quantity of phosphorus is eliminated. Increase of the phosphorus content of the urine indicates an alimentary disturbance. If the urine of an infant who is being naturally fed, though remaining stationary or losing weight, be free from phosphorus, it may be concluded that such an infant is being underfed. Underfeeding lowers the resistance of breast-fed children. The immunity is diminished, infections are more readily acquired, and the course of a disease is likely to be more severe.

Constitutional Anomalies.—Then, too, the constitutional anomalies on the part of the baby sometimes cause a great and very marked influence on the nutrition of the baby. We consider that certain babies are born with certain types of constitutions. We say, for example, that a baby has *exudative diathesis*. What do we mean by that?

VISITOR: That it has eczema.

DR. ABT: What else?

VISITOR: Enlarged glands.

DR. ABT: Enlarged glands, enlarged tonsils; what else?

VISITOR: Enlarged thymus.

DR. ABT: Possibly.

This class of babies is characterized by the tendency to the occurrence of eczema, intertrigo, eczematous patches behind the ears and on the face, the familiar cradle cap on the scalp, and the strophulous eruption on the trunk. Similarly, there is exudation on the mucous membranes. Infants suffering from this constitutional anomaly have pharyngeal catarrh, coryza, angina, glandular swellings in the neck and at the angle of the jaw, middle-ear catarrh, bronchitis, and asthma. In addition, these infants show a tendency to dyspepsia with the occurrence of undigested stools, even when fed exclusively on the breast.

In practice these babies cause endless trouble. The impatient family will frequently insist on a change in feeding; a change of wet nurses rarely gives the desired relief. Recourse to artificial food is likely to lead to greater difficulty. In cases where there is a manifest overfeeding, the food may be reduced to the proper amount. Some of these infants obtain relief by supplementing the breast feeding by cows' milk, which contains a small amount of fat, or by preceding each breast feeding with one-teaspoonful doses of nutrose or plasmon in saline solution. These are casein preparations and supply the deficiency of casein and salts in breast milk.

Well-known clinical anomalies of constitution are observed among prematurely born infants, the offspring of those suffering from organic nervous lesions, possibly the children of tubercular and syphilitic parents, infants suffering from congenital heart disease, and certain types of idiotic children. Such infants are difficult to feed. They may do poorly at the breast or on the bottle. Not the food, but the metabolic functions of the infant are at fault. Who has not seen such infants fail to gain in weight and strength despite every rational method of feeding? The constitutional anomalies of the infant are sufficient to overpower nature's reparative and nutritional provisions. They remain refractory to a greater or less extent to every natural and artificial plan of feeding.

Then we have the babies with *neuropathic diathesis* or neuropathic constitution. These babies are born into the world, and that they are a real entity is just as sure a thing as anything could possibly be in medicine. Neuropathic infants are usually the offspring of nervous parents. The family history relates of tics, or migraine or nervous headache, dyspepsia, neurasthenia or hysteria, or paternal or maternal neuropathic constitution. The infant manifests his symptoms during the first days of life. He is extremely restless, literally wears himself out by his crying and energetic movements; he is awake when he should be asleep, and on account of his restlessness and crying is given food before his digestive apparatus can tolerate it. This infant does not gain in weight. He does not prosper even at the breast; his sleep is not sound; he is easily frightened and jumps at the slightest noise; he is precocious and desires to be entertained. It is difficult to train him to regular habits of sleeping and eating, and on the part of his digestive tract he manifests numerous symptoms, such as colic, flatulency, regurgitation of gas, singultus, and vomiting. The colic of neuropathic origin is the most difficult to manage. The infant himself, distracted, causes pandemonium in the neuropathic family circle, and exhausts the resources of the nurse and the medical attendants.

Advantages of Breast Milk for Sick Infants.—We may speak of another condition in connection with breast feeding, and that refers particularly to feeding sick babies, that is, *detoxication*. An infant who has suffered from a single or repeated insults to the alimentary tract needs, first of all, a short period of starvation. The starvation may be continued for a short period even if water is given. It has become the custom to give weak tea sweetened with saccharin. Experience has taught, however, that while a short period of starvation leads to normal activity of secretion and a disappearance of toxic symptoms in the purely alimentary types of disease, nevertheless, prolonged and continual starvation, particularly in sick infants, may lead to disaster. A catastrophe from starvation occurs more rapidly in an infant whose alimentary tract has been damaged than in an infant who has been previously healthy.

It follows, then, that shortly after a period of starvation has been instituted feeding should be begun. For the purpose of tiding over the sick baby during convalescence following an alimentary disorder, breast milk is the therapeutic agent of the first rank. I may say in passing that in this hospital—and the same is true in other hospitals—we employ three or four wet nurses, and each one of these wet nurses gives milk from breasts and gives us a supply of milk daily. What do we use this for? We use it to feed the very sick babies. A baby comes in here desperately sick. We starve it eighteen or twenty-four hours. Then what shall we feed this baby in order to tide it over? If we give it cows' or goats' milk it is more than likely that the infant will succumb. At the end of twelve or eighteen or twenty-four hours we must give the baby something that seems to have the specific qualities of the species for which it was intended, the specific nutrient quality, the specific antitoxic quality, if you please, and so forth. Now we give this baby breast milk in small quantities. We do not put this baby to the breast and allow it to feed as much as it wants, so that it literally deluges itself with breast milk—because if we did, this baby would develop toxic symptoms. We give it small quantities of breast milk, frequently repeated, and do this for several days, and then by and by he gets a full quota of milk as would be intended for a baby of its age, and it is fed this way sometimes for a week or two weeks even though he makes no gain in weight. The nurses come to us and say that the baby is getting breast milk; it has grown much happier; the temperature has reached a normal level; the skin is pinker and more elastic, but he does not gain in weight. Why not? I want to emphasize this point because it is the principle that we must bear in mind through the whole thought of feeding. Why does not the baby gain in weight?

Professor Arthur Kellar, of Berlin, has very appropriately spoken of this period as the *reparation* period. It is the time when the infant is repairing his damaged tissue and cells before growth and development can take place. During this period, if all goes well, the baby becomes bright and happy, his turgor returns, the tonus of his muscles becomes more normal, his temperature-

curve becomes an unbroken line, he has been lifted out of the abyss into which he sometimes falls, and the gain in weight is the last link in the chain of events which lead from illness through convalescence to health.

Whether the hypothesis is correct or not, it seems to be proved by clinical facts, and for this reason we accept this reparation period as a fact. We recognize the principle that if the baby has been sick and we are changing his diet, we do not expect it at once to gain in weight, but we expect it to repair the damaged tissue and cells before growth and development can take place. That is the point I want to bring out.

Cathartics.—Another question is the use of *cathartics*. If you go among your friends and practitioners who have been practising medicine for some time, if you consult old nurses who have grown gray and weary in the service, and if you talk to mothers of large experience, you will be told that every newborn baby should have castor oil at a certain time, possibly on the third or the fourth day when the milk shoots into the mother's breast, or possibly when the baby is a week old they will tell you to clean the baby out. Now, as a matter of fact, this universal use of cathartics in the newborn babies is perfectly irrational, perfectly nonsensical, but is one of the instinctive things we do; it is founded on tradition; we cannot get away from tradition, and we do these things because they were done for hundreds and hundreds of years and it seems to be the natural thing to do. These babies require no cleaning-out process; as a rule they require no laxative; they simply require food, sometimes a little more food, sometimes a little less food than they are getting, sometimes a slight cessation of food. The long-continued use of cathartic drugs does not improve the condition of the gastro-intestinal mucosa, nor does it diminish the quantity or the quality of toxic products within the alimentary canal, or the cells which provide for the intermediary metabolism. The cleaning-out process which is so long continued acts as an irritant to the bowels, delays detoxication, and renders the prognosis more unfavorable by interfering with repair and the upbuilding processes which must succeed the disease processes and the damage to tissues and cells. The over-

use and abuse of cathartic drugs in the treatment of infantile disorders deserves condemnation as an evil of untold iniquity. What do we do when we give these babies a laxative? You change the entire physiologic function; you increase the motor activity of the stomach and intestines, the food is rushed through more rapidly than it should be, you irritate the cells in the intestinal tract, and more mucus is formed than ordinarily. A few doses of castor oil given to a normal baby will cause the darkest and worst looking stool that you possibly want to see. We can do that with a normal baby, and not only that, we can give the baby laxatives sufficiently long so that blood will appear in the feces. We contend that the use of cathartic drugs is very sadly overdone.

NOTE.—In the September, 1916, number this subject will be continued by Dr. Abt with a Clinic on FEEDING THE NORMAL BABY WITH ARTIFICIAL FOODS.

CONTRIBUTION BY TRUMAN W. BROPHY, M.D., D.D.S., ETC.

ORAL INFECTIONS

Local and Systematic Effects of Infections in the Oral Cavity. Prophylactic and Active Treatment. Good Roentgen Photographs and their Correct Interpretation of Inestimable Value in Diagnosis of Dental Pathology.

It is surprising that the medical profession has not focused its attention on the mouth as a source of general disease, especially in connection with focal infection. The dental profession has realized for many years that blind abscesses about the teeth will frequently produce marked general changes, although their presence may not be noticed by the patient. When we consider that bacteria are present in the mouth at all times, it is not surprising that infections can arise in the oral tissues. The staphylococcus, streptococcus, pneumococcus, and Micrococcus catarrhalis are usually found in all mouths. The number may vary from day to day, and one may be absent on a certain day and present on the following. It is safe to say, however, that the above bacteria are practically always present in the mouths of young children and adults. It does not necessarily follow that an infection takes place. In fact, it happens more frequently that the patient may harbor a micro-organism which is extremely virulent for another individual and yet does not affect him.

The fact that one patient may harbor virulent bacteria and not be affected is explained by the immunization theory. It is also explained by the fact that bacteria rarely gain a foothold on intact mucous membrane. It therefore stands to reason that the person with an unclean mouth is more susceptible to infection than one who has kept his mouth in perfect condition. Caries of the teeth, salivary calculus, and congested gums are fine breeding-

grounds for bacteria. The tissue is thus rendered less able to protect itself against invasion and disease results. The effects may be either local or general.

Considering the number of infections resulting from lesions of the oral cavity, it is somewhat difficult to state definitely what particular portion of the mouth is the most frequent channel of infection. From the dentist's viewpoint, diseases of the teeth would be considered most frequent, while the physician would count the tonsils or other lymphoid tissue the chief avenue of infection. It is safe, perhaps, to state that the tonsils, adenoids, and diseased teeth are the most frequent channels of infection, aside from local injuries.

The local effects when the infection is mild may be slight. The mucous membrane of the mouth, especially that covering the tongue and pharynx, becomes reddened and slightly painful. If this condition continues the salivary secretion is diminished, thus allowing the tongue to become markedly coated and the mouth to have a bad taste. The tonsils may enlarge and protrude into the pharynx. This predisposes to a follicular tonsillitis. The gums may become hypertrophied or atrophied. The irritation of the collection of bacteria between the teeth and gums causes an inflammatory hypertrophy of the gingiva. If the disease is widespread there may be a general hypertrophy. Atrophy is frequently present in dento-alveolitis.

Other local effects about the teeth are abscesses which may produce necrosis of the bone to a greater or less extent. The pus may escape in any of the following ways:

(1) Through the lingual surface and form a sinus at some point not easily observed.

(2) Through the tuberosity of the bone into the pharynx. This opening may not be discovered and a catarrhal condition of the pharynx may be treated indefinitely without a knowledge on the part of the physician of the real character of the disease.

(3) Through the floor of the orbit and form a sinus upon the cheek. Upson describes paralysis of the nerve supplying the eye as a result of infection of the teeth.

(4) Through the antrum of Highmore.

(5) Through the external alveolar plate.

(6) Between the tooth root and socket.

(7) Through the external alveolar plate opposite the apex of the root affected.

(8) Through the cheek at the angle of the jaw.

(9) Into the mandibular canal, thus becoming a source of irritation to the nerve and exciting intense neuralgic pain.

(10) Through the soft parts beneath the chin.

The glands in the neck are frequently involved, especially at the height of infection. These sometimes go on to suppuration and then it becomes necessary to incise them. Other local affections, such as stomatitis, periostitis, sinus infections, inflammation, and infection of the tonsils and pharynx may also be seen.

At the present time the general effects of focal infection have been receiving a great deal of attention, especially from medical men. Volumes have been written, and almost every conceivable form of ache, pain, and deformity have been laid at the door of the teeth and surrounding parts. I am inclined to believe that we are reaching the stage in the furore over the effects of depraved oral conditions upon the general system where we should take count and eliminate much of the unauthenticated conjecture and many of the unproved facts. In other words, we are accusing the oral conditions of causing nearly everything which is unexplainable by any other method. While it is true that ailments such as indefinite joint troubles, gastro-enteritis, and many derangements in the functions of such organs as the liver, kidneys, and, possibly, the spleen, together with the nervous system, may be explained as due to oral sepsis, we must not become overenthusiastic and blame the mouth conditions too often.

Oral sepsis may produce a systemic effect either by direct absorption of the toxic products formed in the mouth or by the ingestion of pus. Rhein[1] has this to say about the effect of ingested pus:

"The effect upon the rest of the body of the constant swallowing of purulent matter has been the subject of much argument.

[1] Dental Cosmos, 1912.

It has been shown that mouth bacteria are carried into the stomach and thence into the intestines. Granting that certain forms of bacteria are destroyed in normal quantities of gastric secretions, the fact remains that the stomach is devoid of acid peptic secretions during a large part of the time. Undoubtedly most of the swallowed bacteria are destroyed not by the digestive secretions of the stomach and intestines, but by competition with the normal intestinal bacterial flora which has, so to speak, pre-empted the ground where nutriment is, after all, limited in quantity.

"When, however, instead of merely swallowing the ordinary bacteria mixed with food, the individual is constantly swallowing certain amounts of pus, an entirely different problem presents itself. The nature, virility, and number of the micro-organisms must have a strong influence as to their ability to overcome the surface immunity of the digestive tract. All forms of diseases of the stomach and intestines are possible. Such bacteria are easily absorbed in the circulation and pass into the lymph-spaces. After this fortress of the body has succumbed, the bacteria, with their poisonous result, appear to be irresistibly drawn to the most vulnerable of the vital organs."

In his work entitled "Insomnia and Nerve Strain," the late Dr. Henry S. Upson has enumerated the following diseases due to dental and oral maladies:

Insomnia.
Insomnia with arterial disease.
Melancholia.
Neurasthenia.
Hysteria minor.
Mania.
Incipient dementia præcox.
Dementia præcox.
Hypomania.

He further states: "Of the viscera responsible for the more obscure cases of nervous and mental derangement I have no hesitation in designating the teeth as the most important. This is not only on account of the common, almost universal, occur-

rence of dental diseases, but because these organs move, during the period of their development, through the solid framework of the jaw, highly enervated and clothed by a membrane sensitive to impact and to corrosive toxins.

"The two most important lesions—impaction and abscess—are in some cases obvious to inspection, but frequently they can only be discovered by skiagraphy. Impactions may be in any region of the jaw. They may be indicated, with some probability, by a gap where the missing tooth should be, but such a gap is by no means conclusive. An extraction may have been made and forgotten, or teeth fail to develop, leaving a gap or a temporary tooth persistent, sometimes for years. Supernumerary teeth may be impacted.

"Many other lesions are potent in causing irritation. Fillings which encroach on the soft tissues or bone are often revealed by the skiagraphs and so remedied. I am unable as yet to give any estimate of the importance of pulp nodules. It is only possible for me at present to make the broad, general statement that irritation and septic poisoning should be removed in every case, and that local results of dental lesions are trifling in comparison with their profounder effect on general health."

Hunter gives the following very lucid classification of possible results of local infection:

"1. Local affections, such as stomatitis, periostitis, sinus infections, inflammation and infection of the tonsils and pharynx. Besides these diseases, we meet with many cases of diseases of the middle ear and different portions of the eye and a host of diseases of the skin often very remote from the mouth. Among these may be mentioned acne, alopecia areata, eczema, erythema, herpes, edema, seborrhea, erysipelas, and others.

"2. Glandular affections, especially about the neck.

"3. Gastric and intestinal affections, such as septic gastritis, septic enteritis, colitis and proctitis, appendicitis, cholecystitis, and septic inflammation complicating typhoid ulceration.

"4. Hematogenous infections, causing pleurisies, empyemata, nephritis, pyelitis, and peninephric abscesses, cholecystitis (and among surgical affections most notably osteomyelitis), and

ulcerative endocarditis, various toxic effects on the blood, nervous system, and kidneys connected with sepsis.

"The poisons act sometimes more on one particular tissue; for example, blood (causing septicemia, pyemia, anemia); or the nervous system (producing severe mental and degenerative effects). Sometimes the toxins may act more on the organs of excretion; for example, the kidney (producing nephritis); sometimes on the skin (producing rashes); sometimes on the joints (producing so-called rheumatic swelling)."

There is not much I can add to the terse classification of Hunter. He has covered the ground very well.

Bunting (Dental Cosmos, 1913), in an article entitled "Effects of Depraved Oral Conditions upon Nutrition," has this to say about children: "In addition to their effect upon the food and oxygenation of the cells, the depraved oral conditions may also affect those other indispensable life conditions, namely, proper physical condition, proper waste removal, and proper innervation. We know full well that among the vast army of children with uncared-for and diseased mouths there is in all of them an incredible amount of infection and fermentation. Much of this infection exists in the cavities of the teeth and about them, whereby all foods that are eaten are contaminated with infectious material. It is possible that in children, especially, the infections which are passed down the digestive tract are killed by the high hydrochloric acid content of the stomach. Regarding this there is some question, as it is found that in individuals having highly infected mouths the intestinal infections are also above normal. This fact is being made use of in the National Jewish Hospital for Tuberculosis in Denver, where the unfailing rule has been established to insist that the mouths of patients be put in perfect condition before beginning specific treatment, and it is claimed that this practice has been of great benefit in the treatment of tuberculosis."

Dr. Nodine, of New York City, in his article, "Scope of the Mouth Hygiene Movement," published in the Dental Brief for July, 1913, said: "When Dr. Keyes can cut down the number of infectious diseases 59 per cent. in the first six months of his dental

service in an institution in Boston having 325 children, and eliminate infectious diseases almost entirely the second year, our hopes are made of more substantial stuff than dreams."

I could continue to quote from the mass of literature at my command, but it seems to me that the opinions expressed above adequately cover the situation and leave little to add.

This is truly a formidable array of facts for a medical man to properly control unless he is versed in the diagnosis of diseases in and about the mouth. It is difficult for our medical students to obtain an adequate knowledge of dental diseases, as the subject is not, with few exceptions, touched upon in medical schools.

The professors and their associate teachers in medical colleges are all specialists. The professor of medicine is an internal medicine specialist. The professor of surgery is a specialist, as his specialty is that field of surgery not covered by the ophthalmologist and otologist, gynecologist, laryngologist, dermatologist, genito-urinary surgeon, etc., and so medicine is divided into many departments of practice, the head of each department paying little attention to the work of the others. The only general practitioner of medicine at the present time is the physician who practices in the country, upon whom the people call for the treatment of all the ills with which they are afflicted, and it is the great majority of mankind that is dependent upon the latter class of medical men for professional advice and care; therefore he should be able to look after all their ills.

I do not advocate teaching the medical student the technic, the art of dentistry, for this he could not learn without adding at least three years to his medical course. I believe, however, he should prepare himself while in college so that he will be able to recognize pathologic conditions of the teeth and those diseases which are associated with diseased teeth and dependent upon them for their origin. Also, if he teaches the families under his care dental prophylaxis he will avert many general diseases.

The medical student should be taught the origin and development of the teeth, their minute anatomy and physiology, the many morbid conditions to which they are subject, the influence of certain diseases of the teeth upon the general health, their part

in forming the initial lesion of trigeminal neuralgia; how to make a correct diagnosis of dental pulpitis, pericementitis, periostitis, and alveolitis; of dental alveolar abscess and its consequences, among which we may name maxillary ostitis, caries and necrosis of bone, empyema of the antrum, injuries and diseases of the terminal branches of the fifth nerve, especially the third or inferior maxillary division, and rhinitis attended with suppuration. He should be taught how to recognize excementosis, pulp nodules, inflammation of the glands, synovitis, myelitis, tumors and cysts of dental origin, etc., etc.

He should become familiar with the local irritations in the mouth, such as result from thermometric changes upon metal fillings in teeth, and thereby recognize a frequent cause of facial neuralgia. He should be taught that artificial dentures, by reason of their undue pressure upon the terminal branches of the fifth nerve, especially the palatine and inferior maxillary nerves as they emerge from the anterior palatine and mental foramina, respectively, are often the cause of extreme neuralgic pain and that medication will be useless while the exciting cause remains. Every dentist of considerable experience knows that patients who wear artificial dentures often complain of the same kind of pain which they experienced before their natural teeth were lost and while they were suffering from pulpitis. They should know the cause of such pain. The medical student should be taught to observe irregularities of the teeth which often cause facial deformities, so that he may advise families who employ him professionally that the dentist's art can correct such defects. He should know the laws of hygiene in the broadest sense, and instruct patients and impress upon them the importance of strictly observing these laws in caring for the teeth of children.

The prevalence of great quantities of pus exuding from around the necks of the teeth, from so-called pyorrhea alveolaris, is constantly depressing the patient's vitality, and the physician should recognize this diseased condition and direct his patients to a dentist competent to employ the necessary treatment indicated in individual cases.

The medical student should be taught how to skilfully treat

fractures of the inferior maxilla; that external bandaging is of no value, often positively detrimental, except as it is used as an auxiliary to interdental splints. Again, in the management of these fractures, he should regard the services of the dental surgeon essential in constructing and adjusting splints to secure most satisfactory results.

If medicine is "the healing art in all its branches," and if, as we believe, diseases of the teeth are more prevalent than any other diseases of the human family, the institutions of medical learning must demand that every student shall possess the same amount of knowledge of these diseases and the diseases associated therewith and dependent upon them for their origin as the student acquires of the diseases of other parts of the body.

I do not wish to be understood as advocating that the physician should always treat the diseases named, but should acquire the same degree of intelligence in regard to their pathogenesis as he does in regard to other abnormalities and direct his patients as he might if they required treatment of the eyes, the skin, or the throat, to the proper practitioner for the service required.

I do not charge the medical faculty with attaching too little importance to oral diseases, but, as I previously stated, each medical college professor is a specialist, and so absorbed is he in the work of his own department he does not realize how extensively, through modern scientific research, the field of the stomatologist has widened and how essential a knowledge of it has become in considering in a general way the ills of mankind.

As the dental college has included in its curriculum such branches of the medical studies as the student of dentistry requires a knowledge of to prepare him for his life work, so too should the medical school teach its students the essentials of dental and oral pathology and surgery to enable them to recognize the various diseases of the mouth with which they come in contact so that the most approved treatment may be employed.

The **treatment of oral infections** is divided into prophylactic and active treatment. *Prophylaxis* should commence with the nursing infant and should never be relaxed. The epithelial surface of an infant's mouth should never be handled roughly, as

infection may take place readily through the denuded membrane. The mouth may be flushed with water, but it is more essential to keep the breast or rubber nipples thoroughly clean. If these details are followed carefully it is noticeable that gastro-intestinal disturbance is practically absent.

During dentition it is essential to keep the teeth clean, and if caries should develop, a dentist should be consulted and the cavity properly sterilized and filled. It is essential that the child be taught to breathe through the nose, as bacteria and dust particles can be handled better there. Anything which interferes with nasal breathing should be corrected.

In an adult the condition of the mouth should be as rigidly cared for, if not more so, than in children. In smokers, especially, and in those who use alcohol it is essential that the teeth be kept thoroughly clean and filled. Filthy mouths have accounted for many cases of pneumonia with death following operation. Just because an operation is an emergency is no good reason why a filthy mouth should not receive at least emergency treatment.

The *active treatment* consists in restoring the parts to normal conditions. The course pursued by the oral surgeon must necessarily depend upon the conditions present. Thus, if the oral infection be a dento-alveolitis or an abscess of a tooth, the treatment should consist in alleviating the infection. When the condition is mild and consists of a carious tooth or salivary calculus, immediate attention should be given to the diseased part in order to prevent further destruction. If taken early, the general symptoms will not have an opportunity to become marked.

If the mouth conditions are severe enough to produce general symptoms, the treatment should be directed toward these. This does not mean, however, that one should neglect the primary cause. It is well to look to the throat and nasopharynx for diseased tonsils and adenoids, especially when the teeth or adjacent parts are not markedly involved. It is inadvisable to attempt to remove tonsils and adenoids when these are acutely infected. They should be treated locally and the patient should receive such general treatment as necessary. This consists usually in active elimination continued over a period of three or four days.

Most of these patients are markedly constipated and it is surprising the relief given them by active cathartics. The use of freshly prepared compound cathartic pills, two or three at night, followed in the morning by a large dose of Epsom salts is especially recommended. The use of salicylic acid preparations will greatly relieve the patient. A gargle should be employed regularly at intervals of at least every two hours.

Logan, in a recent paper, briefly outlines the appropriate procedure to pursue when a patient is suffering from a disease possibly caused by a chronic oral infection. He states: "The appropriate procedure is to have radiograms taken of all the teeth and their supporting structures, and of the jaws for partially or totally impacted teeth. If questionable areas are found, blood examinations should be made (the differential count shows an increase in the polymorphonuclears from about 65 to 73 or 74 per cent. and over). Then, if the physician with the patient will consult the family dentist or oral surgeon to determine if the infection can be controlled with retention of the teeth, or if extraction, with or without curetment, is indicated, and also ascertain the important information whether or not any tooth contains an infected pulp which the radiogram would not reveal in any case, then and then only will the patient's welfare in so far as the mouth and teeth are concerned be properly protected."

Radiography in Dental Pathology. The making of good Roentgen photographs and the correct reading of them is of inestimable value in making a diagnosis. Even the best of skiagraphers often obtain a very poor picture, and I have sent patients back four or five times before securing a skiagraph which clearly outlined the pathologic condition. It does not do to accept a skiagraph unless it is absolutely clear and shows the parts in every detail. On the other hand, even the clearest Roentgen photograph is often misinterpreted.

At a meeting of the Odontological Society of Chicago, February 1, 1916, I spoke as follows:

"I have lately been impressed with the condition known as darkened area in Roentgen photographs not only from the work that I have had done, but on account of reviewing the work of

others. It was not until probably four years ago that I was impressed with the mistakes I had made in reading *x*-ray pictures, and that was brought about in this way (I point this out because I am satisfied that a great many have made and perhaps are making the same mistakes that I did): At that time whenever I saw a darkened area around the apex of a root of a tooth I assumed it

Fig. 10.—In this skiagraph a molar and bicuspid are pulpless. One root of the molar has been well filled. The other two were not completely filled. The bicuspid roots are only partially filled, as the skiagraph shows. The woman suffered intense neuralgic pain and was advised by physicians to have the two teeth removed, the pulps of which had been destroyed. The teeth were firm in their sockets, not sensitive to pressure, and while the skiagraph shows darkened area beyond the roots, the bone is not destroyed. The bone about the apices of the roots is normal. Neither the teeth nor the surrounding bone was diseased; the pericementum was in normal condition and the cementum was not affected. A number of expert skiagraphers examined this Roentgen photograph and failed to observe the real cause of the neuralgia. In a good light I found a pulp nodule in the second molar tooth, just above the filling that is in the wall of the tooth. I directed that the tooth be opened, the pulp destroyed, and the nodule removed. This was done, and the woman, who had suffered for years, was permanently cured. The error of sacrificing the bicuspid and molar teeth would have caused the patient a great loss.

meant the presence of an abscess, but when I made *x*-ray pictures of fracture of the mandible I found a well-defined line of fracture, for which I made use of splints, readjusted the fragments, restored the normal occlusion of the teeth, waited six or eight weeks, and then made another skiagraph of the same bone at the same place; I found exactly the same appearance that I found when I had the first skiagraph made, and concluded, before

I learned better, that a fracture still existed. The newly formed bone was not sufficiently dense in structure, not sufficiently ossified to present the same appearance that the normal older bone did, so I was misled. Leaving the splints on for five or six weeks longer and again making a Roentgen photograph, I still found a well-defined line at the fracture; but feeling satisfied that the bone was united, I removed the splints and found that the bone had firmly united and was all right, yet at that time it showed exactly the same condition that it did when the first skiagraph was made. This led me to believe that I had been making mistakes regarding the darkened areas at the ends of the roots of teeth.

"Now I am convinced that these darkened areas we see on the plate are by no means evidence of an abscess. They are evidence, however, that an abscess once existed, that the bone was destroyed or absorbed by pressure, and that perhaps there was infection of the organic matter of the bone to such an extent that some of the bone itself became disintegrated. In some instances we all know a great deal of the bone will become disintegrated and large cavities will be left at the ends of the roots of the teeth; but these large cavities, everything being done to exclude further infection by having the root well-defined, will be filled by new bone.

"In this connection I would like to state that at the meeting of the Clinical Congress of Surgeons of North America, held in Boston last fall, Dr. M. L. Rhein read a paper on this subject and presented, by means of the stereopticon, some plates showing teeth the roots of which were not well filled, with rather large areas of destruction at their apices (Fig. 11). Then he showed the same teeth, skiagraphs of which were made two years later (Fig. 12). He had removed the fillings from the roots of those teeth and refilled them, following which there was sclerosis, the bone was well developed, completely surrounding the end of the roots, and showing as complete an alveolus as surrounded any of the teeth with living pulps. The darkened cavities were filled with new bone.

"All we need in the treatment of such teeth is asepsis. We must have these roots thoroughly cleaned, sterilized, and well filled. If so, the abscess cavity which is shown so clearly and

readily on these plates will take care of itself. Sometimes we may be obliged to perforate and evacuate the fluid from it, but if the cavity can be thoroughly sterilized by treating the tooth root

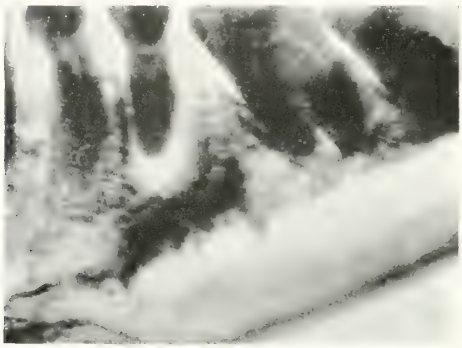


Fig. 11.—Showing teeth the roots of which were not well filled, with rather large areas of destruction at their apices (M. L. Rhein).

or through the alveolar process, it will, if well drained, take care of itself. I have no more reason to believe that such a tooth should be extracted than I would believe a patient who had osteo-

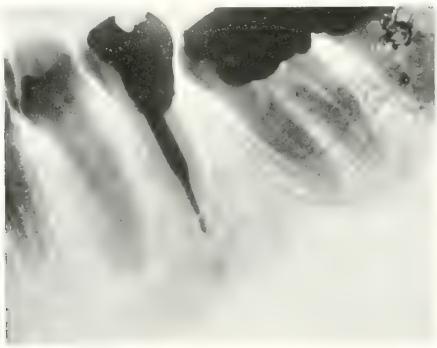


Fig. 12.—The same teeth shown in Fig. 11 two years later, showing bone regeneration (M. L. Rhein).

myelitis of the radius or ulna should have his arm amputated. The principle is precisely the same, and if we can convince our medical friends who are laboring so hard with this subject that

disease of the character that has been presented here in these plates is amenable to successful treatment, and that the sacrifice of such teeth is not necessary, we will accomplish a great work.

"There has been and there still is a tendency to remove teeth because their diseases are not understood. There is too much of a tendency to make a clean sweep and remove diseased teeth as well as those that are not diseased. If a tooth is made firm by putting on bands and splints, after getting rid of the infection, it

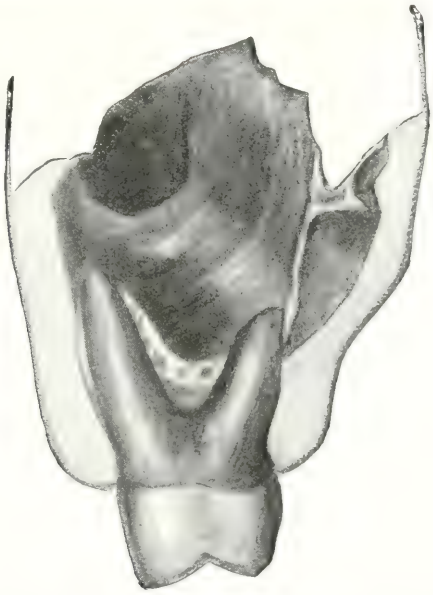


Fig. 13.—Illustration showing bicuspid root projecting into the antrum (Conboy, *Dental Cosmos*).

can be preserved and made to serve the purpose for which it was intended many, many years. Roentgen photographs are of inestimable value in the examination of teeth the alveoli of which have been almost wholly lost, and the teeth retained only by membranes. Such teeth stand in very shallow alveoli. They have so little support that they move with the slightest pressure. In these cases a skiagraph is of value in pointing out loss of bone and to convince us that it is absolutely necessary to remove the

teeth or tooth the alveolar processes of which have been destroyed by disease. I would be loathe to accept the statement that a new alveolar process may be developed or built up with proper treatment until I had seen at least half a dozen or a dozen positive demonstrations of that kind.

"There is much to be said on the subject of skiagraphy. Let me call your attention to a specimen: Here is a specimen in which one-third of the root of the tooth stands up in the antrum (Fig. 13).

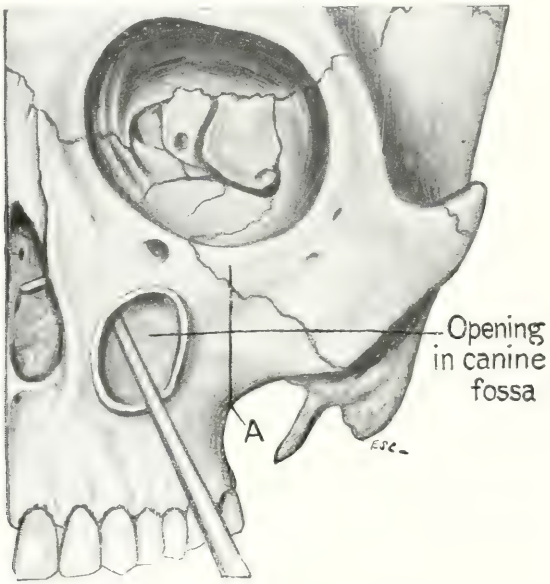


Fig. 14.—Large opening through the canine fossa for the treatment of chronic empyema of the antrum. The instrument is introduced to dilate the osteum maxillare (Brophy, *Oral Surgery*).

That tooth is not in bone at the end of the root at all. If an abscess should form there, pus would escape into the antrum, of course. Our friends, the rhinologists, make great mistakes in overlooking this condition, because at least 75 per cent. of all infections of the antrum of Highmore come through the medium of diseased teeth rather than through the nose. The disease may come from nasal trouble and from the frontal sinus and ethmoid cells, but the teeth are more diseased than any other part,

and abscesses form about their roots; even an abscess of a central incisor may involve the antrum by the pus burrowing through the cancellated bony tissue. When this occurs, a generous opening through the canine fossa should be made, in chronic cases, so that the antrum may be explored, an ocular examination made of all its walls, the diseased tissue removed, and the antrum restored to health without the loss of any tissue that would be

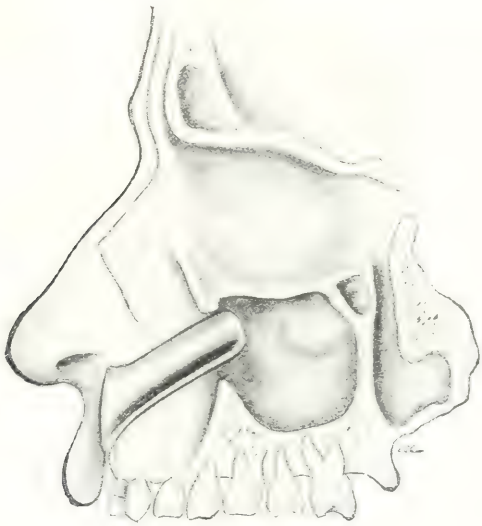


Fig. 15.—Showing the manner of introducing a gutta-percha plug in the antrum. This maintains the opening through which ocular examinations may be made and the antrum treated from time to time. The cavity may also be illuminated through this opening with a small incandescent light (Brophy, *Oral Surgery*).

detrimental to the patient." I here present a group of illustrations which have direct bearing upon the subject of oral infections.

At the same meeting Dr. Oscar A. Strauss, of Milwaukee, spoke as follows:

"I greatly appreciate the importance of radiography and its value in dental pathology.

"During the past two years I have treated many cases of rheumatism and arthritis in various forms caused by infection—mostly by infections of the mouth. About six months ago I had

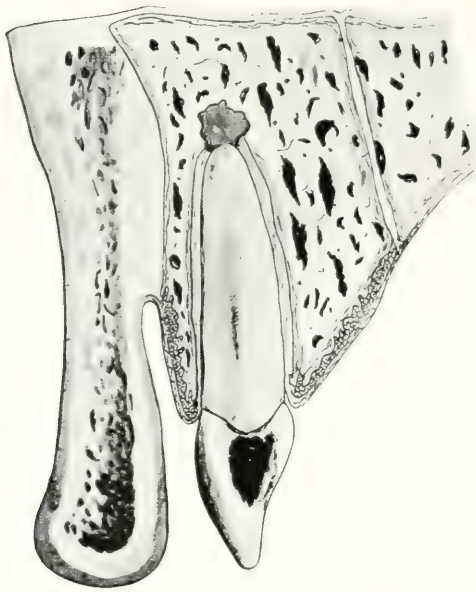


Fig. 16.—Upper incisor tooth showing apical abscess. These abscesses or any irritation to the dental alveoli from any cause whatever will result in dento-alveolitis (Brophy, *Oral Surgery*).

Fig. 17.—Acute dento-alveolar abscess in maxilla which penetrated the antrum and also involved the orbit (Oakman).

a patient who came to me with a bad arthritis deformans—he was barely able to walk for two years, and was practically unable

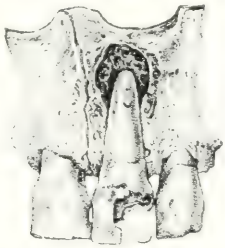


Fig. 18.—Cavity produced in the maxilla by an abscess at the end of the root of the left central incisor. The crown has been destroyed by caries and the canal of the root is open. The external wall of the alveolus is wanting (Heider).

to dress himself. The patient was a man of means and was able to consult many prominent physicians. He had visited sanato-

Fig. 19.—Dento-alveolar abscess at the root of an upper incisor tooth discharging into the nose (Brophy, *Oral Surgery*).

riums, hot springs, mud baths, etc.; in fact, he tried everything to eliminate the uric acid condition. He came to Chicago, saw the most noted physicians of this city, and they were unable to

Fig. 20.—Abscess of an upper cuspid tooth discharging through the bone just beneath the orbit and upon the cheek. (Brophy, *Oral Surgery*).

Fig. 21.—Alveolar abscess discharging on the face, leaving an unsightly scar (Brophy, *Oral Surgery*).

find a focal infection; finally he came to me, and I examined him thoroughly. I found nothing; however, I started treating him, and looked again and again. I was rewarded by finding an infected pocket back of the third molar. An autogenous vaccine was made of this pocket and after a few injections his hands cleared up. Then a pustule appeared in the left knee, which was

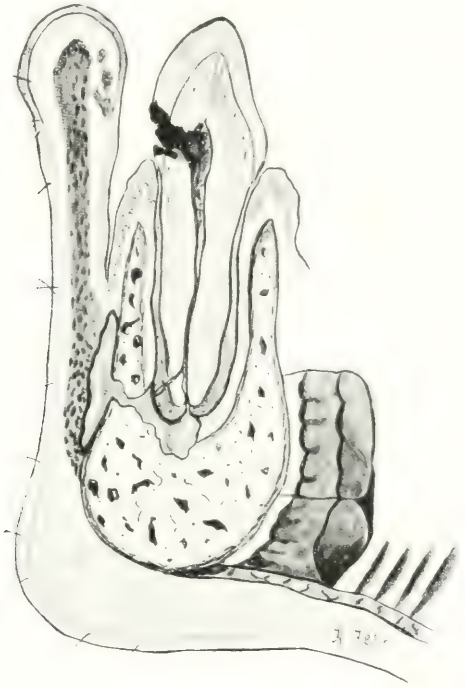


Fig. 22.—Acute alveolar abscess of a lower cuspid tooth with pus cavity between the bone and periosteum (Brophy, *Oral Surgery*).

badly infected. A specimen of the pus was taken from this pustule and compared with the pus taken from the tooth pocket—they were of the same type. The autogenous vaccine cleared up the condition in six weeks' time.

"Last week I discharged a patient who had not walked for four years; she was a very heavy woman, weighing 215 pounds, and was about 5 feet 5 inches tall. The patient had a blood-

pressure of 240. In this case radium was resorted to until the infection was found. I discovered in one of the infected joints a little pus containing a Pneumococcus fusiformis. For some time I was unable to locate the infection, until finally I discovered through radiography that the lower anterior teeth were infected; an autogenous vaccine was made of this, and very favorable results were obtained.

Fig. 23.—Lower tooth showing chronic dento-alveolar abscess, discharging on the face under the chin. The pus sometimes passes directly through the substance of the mandible, discharging immediately beneath the chin (Brophy, *Oral Surgery*).

"I cite these cases merely to demonstrate the importance of radiography in dentistry. These patients had been the rounds of both physicians and dentists. It also shows that one cannot depend alone upon the color of the tissues surrounding the teeth. Radiograms should be made for a positive result. On the other hand, we must be very careful not to extract too freely—as pointed out by Dr. Brophy—simply because there is a slight discoloration

in the radiograph. I have seen patients who were advised to have their teeth extracted — patients with excellent teeth and absolutely no pathologic condition existing. Therefore it is not only necessary to have radiographs made, but also to be able to interpret them properly."

Fig. 24.—Dento-alveolar abscess discharging above the thyroid cartilage. Such sinuses are always misleading. They may discharge direct from the glands or from infection of the bone, but more frequently dental abscesses (Brophy, *Oral Surgery*).

Dr. Geo. J. Dennis, specialist in diseases of the throat, nose, and ear, who was present at this meeting, made the following remarks:

"About the only thing I have to say is to offer a protest against the indiscriminate removal of teeth which is going on at the present time.

"It has been my pleasure to see quite a number of skiagraphs of teeth that have been passed upon by physicians in which they distinctly stated the teeth should be removed. For some reason

the skiagraphs were brought to me for interpretation, and after looking over them I do not hesitate to say that in about 75 per cent. of the cases I could find very little, if anything, in these skiagraphs which would point to the necessity for the removal of those teeth.

"I have in mind one case, a patient of one of the gentlemen present, in whom fifteen teeth were advised to be removed from

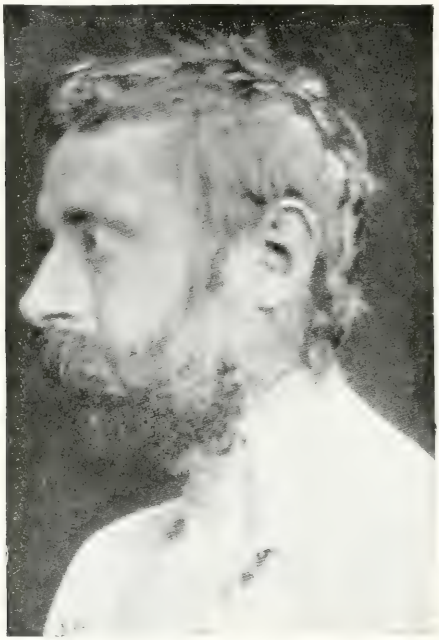


Fig. 25.—Abscess of the third molar with a fistula opening in the sternal region (Schröder).

the mouth of a man who was a little over forty years of age. Of those fifteen teeth, skiagraphs of which had been made, there were only four that I could say were at all suspicious. Of those four, there was some doubt as to the necessity of their removal.

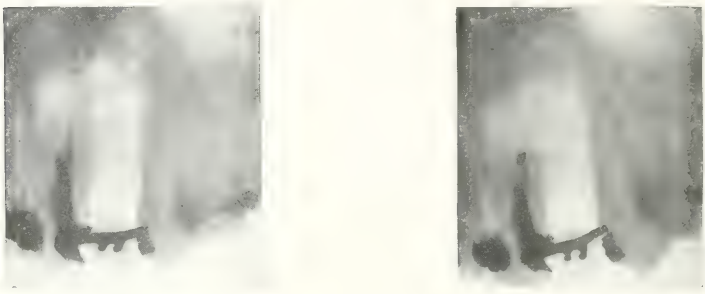
"There is no doubt that focal infection has quite an important bearing upon the general physical condition of these patients. However, it seems to me that it is a fad very largely that is being

much overdone, and I believe there will be a great reaction before long. Just how soon that reaction will occur I do not know. I

Fig. 26.—A sinus leading from the infected first right inferior molar through the external plate of the bone beneath the skin to a point in the sternum opposite the third rib. Bismuth paste shows the tract of the sinus and also a large carious cavity in the sternum (Deichnieller).

think many of the men who have laid the greatest stress upon the subject of focal infections are beginning to see some of their fail-

ures, and there are quite a number of failures. There are so many points about the body besides the teeth and throat—and



A *B*

Fig. 27.—Apical abscess on central tooth of long standing, necessitating amputation of the root (Brophy, *Oral Surgery*).

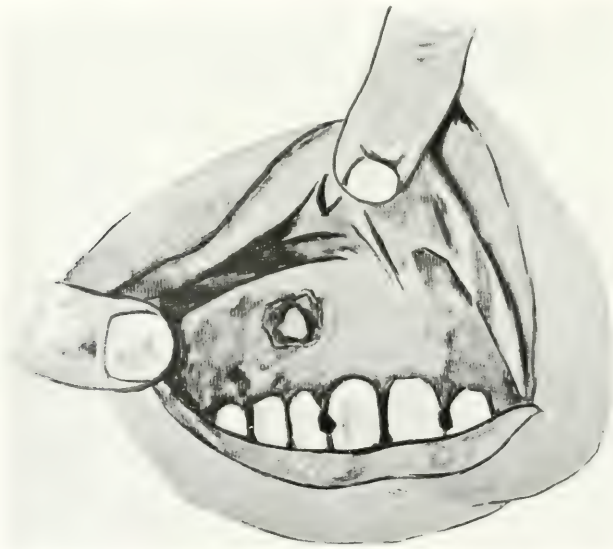


Fig. 28.—Showing preparation for excision of root. Diseased condition caused by dento-alveolar abscess (Brophy, *Oral Surgery*).

the throat, nose, and ear interest me particularly—that the question comes up as to whether they have been successful in finding the real foci of infection. It is a thing I believe we ought

to be conservative about, and yet there is no doubt about the truth of a great many of their findings. We all see, I think, remarkable recoveries of patients after the discovery of some focal infection and its removal. I have no doubt but what we will find many more if we are successful enough in looking for and finding them. I do not wish to be understood as belittling in any

Fig. 29.—Incorrect method of operating for the treatment of fistulæ resulting from dento-alveolar abscess (Brophy, *Oral Surgery*).

degree efforts in that direction; at the same time I want to make a plea for a little more conservatism in reference to the extraction of teeth."

It is apparent that the condition of human suffering calls for a more thorough pathologic training. I believe that errors in diagnosis that I have pointed out are largely due to the fact that medical professors have not until recently been impressed with

the importance of teaching students dental pathology, and furthermore, that dental college teachers have not dwelt at sufficient length upon the subject of general pathology.

On the one hand, it is extremely difficult to impress upon the teaching faculty the great importance of teaching subjects which were not taught when they were students, and a knowledge of

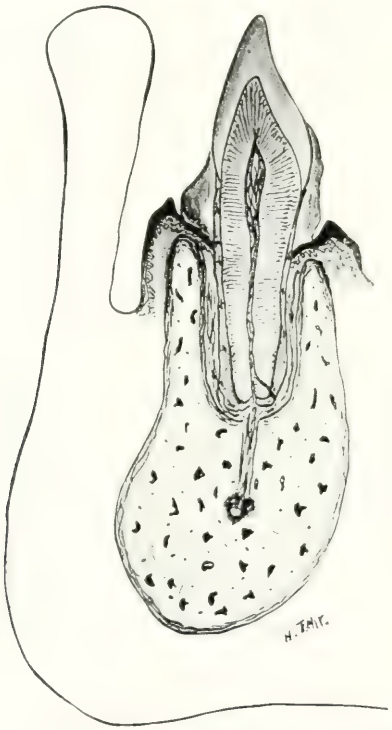


Fig. 30.—Lower lateral tooth showing deposit of salivary calculus, causing inflammation of the gum (Brophy, *Oral Surgery*).

which they have never had an opportunity to acquire. The early dental college curriculum gave a very meager course in dental pathology; today the curriculum has been so expanded that the subject of general pathology is not an important part of the course of study. The pathologic laboratories teach the etiology of disease and its many manifestations. We expect, in view of the

growing demand for a more general knowledge of the infections of oral origin, that all well-equipped medical colleges will take this subject up and teach it in its fulness.

With these improvements in education the physician and dentist of the future, by co-operating in the diagnosis of complicated cases, will be more likely to make correct diagnoses. Otherwise, errors in diagnosis and treatment of patients will probably occur as frequently as they do now unless the physician, the dentist, and the radiographer co-operate in diagnosis.

CLINIC OF DR. CHARLES SPENCER WILLIAMSON

COOK COUNTY HOSPITAL

A MEDIASTINAL TUMOR—PROBABLY HODGKIN'S DISEASE

Rapid Enlargement of Cervical Glands. Subsequent Development of Hydropneumothorax.

Present History.—This patient, forty-five years of age, single, came into the hospital on the throat service on February 17th, complaining of sore throat, swelling of the neck, headache, loss of weight and strength, poor appetite, and especially loss of sleep. He had a marked dryness of the throat, a cough which was practically incessant, and at times was so severe that he vomited. He has had night-sweats and, he thinks, some fever. Five months before entrance he first began to complain of a sore throat, and following this he noticed a few lumps just below the right sternoclavicular articulation and a few more just above the clavicle in the posterior cervical chain. In addition to this he has another symptom which is interesting, and that is an intolerable itching of the legs. There is no history of tuberculosis or carcinoma in the family. He had an initial lesion of lues followed by a rash about three years ago. He had gonorrhea two or three times. He smoked ten or twelve cigars a day for years, but stopped several months ago for he found that it was not good for him. He was treated for syphilis with mercurial tablets and had one injection of salvarsan.

Examination in the nose and throat department showed that the septum deviated a little to the left. The neck showed a large number of small discrete glands around the left side. He had no difficulty in swallowing. The glands were all movable and two

were taken out for diagnosis. The respiratory excursion showed no change. There was some dulness over the upper chest, anteriorly, on the right side. It soon became evident that the case should be transferred to the medical department, and he came

Fig. 31.—Mediastinal tumor. *X*-ray plate early in disease. Masses of glands outlined approximately.

under our care on February 21st. Since then he has had little or no increase of temperature. The first blood-count showed 34,100 leukocytes; a second count showed 34,000. There were $80\frac{1}{2}$ per cent. polymorphonuclears, $5\frac{1}{2}$ per cent. large mononu-

clears, 2 per cent. small mononuclears, 1½ per cent. eosinophils, and 1½ per cent. basophils.

He expectorates a little; his cough has been very bad, but is better at present. Examination of the sputum revealed only a few elastic fibers and no tubercle bacilli. Another specimen of sputum examined by the antiformin method was negative. The urine showed no abnormal findings.

In view of the fact that he has been treated for lues a Wassermann was done and the blood-serum was reported negative.

Physical Findings.—When he was referred to our service we made a complete examination, and I will give you the findings as we then found them. They agree very well with the present findings. The cervical glands have grown larger and are now as large as marbles. The chest is symmetric; expansion is fair. There are some dilated veins on the chest; you will notice in particular the dilatation of the veins running over the shoulder.

The apex-beat is in the fifth intercostal space 10 cm. from the midsternal line. There is an area of dulness which is difficult to describe. On percussion the second intercostal space on the right side is distinctly dull; the whole upper portion of the chest is flat, or so very dull that it is practically flat, and all over this area we hear distant breathing which is slightly tubular. We do not feel any abnormal pulsation. This is very important because of the possibility of an aneurysm. In an aneurysm which occupied the whole upper portion of the chest we would have an enormous pulsation. There is no difference between the two radials or between the two carotids; there is no delay of the pulse. If we look at the *x*-ray (exhibiting *x*-ray plate) we will see that the apex of the heart is pushed out a little bit, and that the right side shows a big shadow which is well marked in the upper portion and very well marked in the lower. The densest part of the shadow is in the region of the third or fourth rib and extends down to about the fifth rib, where the greater amount of dulness is. We also see the shadow at the area corresponding to the base of the great vessels. The outline of the shadow is irregular, not round, and that generally means that it represents a solid mass in contradistinction to an aneurysm.

The finding of a large area of dulness in the right side of the mediastinum, without any pulsation, is not compatible with a diagnosis of an aneurysm. Then, too, the glands in the neck, which are growing larger all the time, add to this certainty, so that there is very little doubt that there is a solid tumor in the chest, probably a mass of glands.

In the abdomen we find no particular tenderness, but a little rigidity over the liver. An interesting finding is the distinctly enlarged spleen. The femoral and the inguinal glands are twice the size of a bean. The epitrochlears are not enlarged.

Where is the principal lesion located? Undoubtedly it is in the mediastinum itself. The other findings are probably secondary, because as we go down toward the bottom of the neck we find larger glands, and the natural assumption is that those in the mediastinum are still larger.

Diagnosis.—What is the nature of the tumor? There is an increase in the leukocyte count and an increase in the size of the spleen. There is nothing pointing toward tuberculosis, and I think that it can be ruled out with practical certainty. We have excised two of these glands, but the report was not conclusive. The present physical findings can hardly be explained by a luetic infection. The Wassermann is negative at present; he had some treatment two or three years ago, and if he had mercurials and iodids he would not be likely to develop these symptoms. On the other hand, it might be a sarcoma, but then we would have great difficulty in explaining the definitely enlarged spleen. Besides, there is no infiltration of the periglandular tissues. We have one form of semi-malignant tumor, the so-called Hodgkin's disease, which fits in well with this picture. I believe this to be that peculiar type of the disease which starts in the mediastinum and afterward involves the other glands. I think the term "Hodgkin's" is applied to a great many conditions in a rather loose way, and yet there are some typical cases. In the ordinary type there is a large collar of glands around the neck. Then we have a type where the glands in the abdomen enlarge first. Occasionally we see a case which starts in the mediastinum and has a peculiar, recurrent temperature. This case represents the

type of Hodgkin's disease which begins in the mediastinum, although it has not shown the peculiar temperature-curve. So the probability is that this is a Hodgkin's disease affecting all the glands in the body, with an enlarged spleen.

An interesting feature is the frequency with which mediastinal disease simulates disease of the larynx. When this patient entered the hospital he was thought to have some laryngeal trouble, and it was only after careful examination in the nose and throat department that this was found to be a secondary matter. The so-called brassy cough should always make one think of mediastinal disease, a condition that is very easily overlooked. I remember a patient on whom a diagnosis of gall-bladder disease was made by one of our most experienced clinicians because of a peculiar temperature, and who was about to be operated upon. I suggested an *x*-ray examination of the mediastinum, which revealed a mediastinal tumor of just this sort.

The **prognosis** is self-evident: the condition is almost necessarily fatal. The only chance for this patient to get well is that I am mistaken in my diagnosis and that the condition is syphilitic. He is on very active antispecific treatment. He is getting mercury and iodid of potassium in large doses, but without any apparent effect.

Additional Note, April 19, 1916.—This patient with the mediastinal tumor whom I showed you several weeks ago has, up to two days ago, remained in pretty much the same condition, except that the glands have been growing steadily larger, in the cervical and axillary regions especially. They are now the size of pigeon eggs. The spleen shows no change and can be felt two fingerbreadths below the costal arch. Two days ago the patient suddenly complained of intense pain on the right side, and has had several severe attacks of coughing. Inasmuch as he has had such attacks for a long time, little attention was paid to it for some hours, until it was noticed that he was intensely dyspneic. A careful examination of the chest elicited the following points: The entire right side of the chest gave on percussion a deep, drum-like note. The liver dulness was replaced by a resonant note; the lower margin of the liver extended down a little below the

umbilicus. The apex-beat of the heart was displaced to a point 1 inch to the left of the nipple line. The temperature was normal, pulse 120, respiration 48 per minute.

These findings were, of course, characteristic of *pneumothorax*. Further examination, by auscultation, gave the characteristic amphoric sound with absence of breath sounds over other areas, and the coin test confirmed this. These findings are still unchanged, the patient is still intensely dyspneic and suffering greatly.

We must now ask ourselves what led to the development of this pneumothorax. We should not forget that the patient has been for a long time having intense paroxysms of coughing, and the question arises as to whether or not this alone furnishes an adequate cause for the rupture of some of the air vesicles and the development of pneumothorax. While this is not a common event, there have been a number of cases of this sort, and I personally know of one other. On the other hand, when we reflect on the conditions existing in the mediastinum, and when the peripheral glands are steadily enlarging, it is not at all farfetched to assume that there may be a development of tumor masses in the lung itself and that one of these may have ulcerated through, possibly aided by a paroxysm of coughing, and thus caused the pneumothorax. In either event we may say that there has been a rupture of some of the air vesicles either spontaneously or as a result of the destruction of tissue by a metastasis.

The diagnosis of pneumothorax being made, the next question is, what treatment we shall institute. Symptomatically we have given him morphin, which has made him somewhat more comfortable. The collapse of the lung is the best thing which can happen to the patient, inasmuch as it allows the torn edges to become approximated, and thus gives them the best possible chance of uniting. The chances of this happening would be very much less in case the condition is due to a metastasis.

I have just stated what I believe to be the two likely causes of this pneumothorax, and it may be that some of you are wondering why I have not considered the rupture of a small tubercular cavity as one of the possibilities, especially as this is the commonest cause of pneumothorax in general. I have not over-

looked this point for a moment, especially since we must never lose sight of the fact that there are cases of generalized tubercular adenitis resembling Hodgkin's disease to a nicety. For all this, the most painstaking search has failed to show tubercle bacilli

Fig. 32.—*X*-ray showing mediastinal tumor after development of pneumothorax.

in the sputum, and up to the present our inoculation experiments have been negative, so I am obliged to exclude this source.

The diagnosis of pneumothorax in this case is so clear that it scarcely needs any further verification. On the other hand, it is always wise to substantiate the diagnosis in every way possible.

so that I have just had an *x*-ray plate of the chest taken and, as you will see, it gives the classical picture of pneumothorax. It is very instructive to compare the two plates, one taken some weeks ago showing the mediastinal mass, and the other taken yesterday showing the pneumothorax.

Additional Note, April 26th.—I am showing you this patient for the third time today because his condition has changed again. The pneumothorax is still present and, as might have been anticipated, a small amount of fluid has developed in the chest, so that he has a typical *hydropneumothorax*. When you take hold of the patient and shake him gently, with your ear over the back of the chest, you can hear the succussion sound in the clearest manner. In regard to the nature of the fluid, it is probable that this is of a serous character, since the temperature has not gone up materially and the other evidences of pus have not developed. The amount of the fluid is small, hardly enough to be demonstrated by percusssion, but readily demonstrable by the splashing elicited on succussion. This small amount of fluid develops in most cases of pneumothorax and, in my judgment, does not add materially to the gravity of the condition.

Since the last time I showed you this patient we have succeeded in adding somewhat to the accuracy of the diagnosis of Hodgkin's disease. When the patient first entered the hospital we extirpated a small gland underneath the clavicle, and a careful histologic examination of this showed nothing at all characteristic, the specimen looking more like a simple adenitis. About ten days ago we extirpated one of the larger glands, perhaps the size of a pigeon egg, from the cervical region, and I have just received the pathologic report. This is to the effect that it is certainly not tubercular, and while not absolutely typical, is sufficiently so to warrant the histologic diagnosis of Hodgkin's disease. As you know, this disease is at the present time being made the subject of a great deal of discussion, which it would hardly be profitable for us to review in a clinic of this nature. It is well, however, to notice that the disease, whatever its etiology may prove to be, attacks the entire lymphatic apparatus, so that in this sense it is a systemic disease of the lymphatic tissues of the body including, as in this case, the spleen.

MULTIPLE TUBERCULAR SEROSITIS

Demonstration of Tubercle Bacilli from Both Pleura and Peritoneum.

THIS patient is an American, thirty years of age, who came into the hospital on May 1st with the following rather interesting history: He was in this hospital six weeks ago with a pleurisy with effusion. While here $1\frac{1}{2}$ liters of fluid were removed from his chest by pleural puncture. He left the hospital with the pleural condition subsided and the fluid had not recurred.

His present complaint is that his abdomen is enlarging. He has pain in that region and has lost 12 pounds in weight in six weeks. He considered himself well until about two weeks ago when the pains in the abdomen started and the swelling appeared. This swelling has lasted fifteen or sixteen days, but the larger part of it has occurred in about a week. The pain is on both sides of the abdomen, especially in the flanks, and during the last week has been pretty severe. It is cramp-like in character and occurs every two or three hours. He says he has never had any similar pain. He has been quite alarmed about the loss of weight. A year ago he weighed 178 and now he weighs about 115 pounds. The bowels usually move twice a day. He has no cough, no night-sweats, and no anorexia.

Physical Examination.—General inspection reveals a young man who is rather emaciated and looks quite sick. The thing that strikes your attention first is the emaciated chest and the markedly distended abdomen.

The *head* and *neck* show nothing abnormal. The left pupil shows slight change due to an injury to the eye a long while ago. The throat is negative, the teeth and gums fair.

The Chest.—The lower aperture of the chest is distended owing to the very greatly enlarged abdomen. Every rib stands out very prominently. There is no particular difference between the two sides.

The Lungs.—Percussion anteriorly shows a slight dulness beginning at the fourth rib and flatness from the fifth rib down. Posteriorly from about an inch above the angle of the scapula down there is flatness. When we auscultate we find both the breath and voice sounds are very distinct, so there are evidences of fluid in the right side. The reason I say this is because the flatness is much too high and much too intense to be caused by the pushing up of the liver. On the left side posteriorly we can hear a rather extensive pleural rub. To be positive that there was fluid we inserted a needle into the right pleural cavity and took out about 750 c.c. of moderately turbid, greenish-yellow fluid which showed 90 per cent. polymorphonuclears, 10 per cent. lymphocytes, and an occasional blood-cell. The specific gravity was 1017. *Tubercle bacilli* were sparingly present. No other organisms were found.

The Abdomen.—The distention has now subsided materially. We took out 4000 c.c. of fluid yesterday and there are perhaps 8 liters still remaining. Examining the abdomen more in detail, one sees that it is a typical frog belly. It overlaps the flanks on the side, the navel protrudes markedly. On percussion we find a little zone up in the center which is tympanitic. All over the flanks, in the pubes, and higher up the percussion note is flat. When we tap the abdomen we get a characteristic fluctuation wave. Auscultation should always be carefully done in these cases; if there is not much fluid you can very often hear a friction rub better than you can feel it. The left flank lower down is very tender, and here and there all over the abdomen we find these tender spots.

The fluid from the abdomen is a little more greenish yellow and is decidedly clearer than that from the chest. The specific gravity is 1015; the cell count shows 85 per cent. lymphocytes and 15 per cent. polymorphonuclears.

Diagnosis.—We must carefully analyze this case in order to arrive at a diagnosis. This young man came in with the history of a pleurisy. A thoracentesis was done and approximately 1500 c.c. of fluid removed, and he then left the hospital. He has not coughed at all. The diagnosis was made of a pleurisy

with effusion, probably tubercular. Very soon he returned with a somewhat increased temperature—the first day 99.2° F., the next day 100.6° F., last night 102° F., and with the abdomen full of fluid. The natural inference is that he now has, in addition to the tubercular pleurisy, a tubercular peritonitis. The onset with fever, the presence of considerable fluid, and the peritoneal rub point clearly to a tubercular peritonitis. The pleural membranes have become involved successively without any involvement of the lungs that we can discover, with fluid on the right side and with a dry pleurisy on the left side.

The diagnosis in this case has been confirmed by finding the organism in the fluid by the Löffler method. (Showing specimen with tubercle bacilli.) We examine for this organism in all cases of supposed tubercular exudates, and you can see as pretty a specimen as you want to see right here. This renders the diagnosis absolutely positive.

Tubercular pleurisy is frequently seen. Tubercular peritonitis is not so common, and the combination of the two is very unusual. In this case the left pleura is also involved, although there is no fluid there at the present time. There is, therefore, an involvement of the three great serous membranes.

You will notice that I have said nothing about the heart. When I first saw him there was marked dulness on the right side. If this dulness had been caused by the liver being pushed up, the heart should be in approximately the normal place, or a little to the left, but on examination we found the heart *greatly* displaced to the left. The apex-beat was out beyond the nipple, but there was no enlargement of the heart, so the evidence was conclusive that the heart was pushed out by the effusion in the right side of the chest. Remember that if the liver is simply pushed up, the heart will not be very materially out of place, but if there is much pleural effusion the heart will be greatly displaced. My judgment is borne out by the fact that since we have removed 750 c.c. of fluid the heart is fully an inch further in. It is difficult to make out the outlines of the heart when the right side of the chest is involved and the heart is displaced, so we have to depend largely upon auscultation to determine the presence of a pericar-

dial exudate. In the absence of any unusual findings I am inclined to think that the pericardium is not involved.

The **diagnosis** is tubercular peritonitis with effusion, tubercular pleurisy with effusion on the right side and a dry pleurisy on the left. The friction rub on the left side is now so marked that it can be felt very plainly. It extends over an area two or three times as large as the hand, so there is no doubt in my mind that he has a rather extensive dry pleurisy on the left side.

This case shows how a patient may be discharged as apparently cured, and yet the focus of infection is still there and later breaks out in another spot. Tuberculosis especially has a tendency to do this.

A last word in reference to finding the tubercle bacilli in pleural and peritoneal exudates. The books will tell you that it is extremely difficult, but with the Löffler antiformin method you will find them very frequently, and it is a much quicker method of confirming the diagnosis than by animal inoculation. If you will try the Löffler method you will find tubercle bacilli in quite a large percentage of cases.

I want to call your attention to the two fluids obtained by aspiration. From the chest we obtained a turbid fluid with a specific gravity of 1017, showing tubercle bacilli in small numbers. The cell count was highly unexpected, in that we found *90 per cent. polymorphonuclears*. I hardly know how to account for this except that possibly there was a low-grade secondary infection previously present. At the time we made the puncture we found no other organisms. The peritoneal fluid showed 85 per cent. lymphocytes, which is about what we might expect.

These cases in which several of the serous membranes are involved by themselves in a tubercular process are not very common. Tubercular pleurisy is by far the most common by itself. Multiple tubercular serositis is very different from the mere studding of the serous membranes with tubercles, such as one sees in an acute miliary tuberculosis. In these cases of serositis there is no generalized miliary process and recovery may and does occur, whereas the acute miliary form is fatal. The diagnosis, as a rule, offers no especial difficulty if one is aware that a multiple serositis of tubercular nature exists.

A CASE OF BRONCHIECTASIS (UNILATERAL)

Long History of Putrid Sputum and Cough, with Acute Exacerbations. Interpretation of Physical Findings. Bronchiectasis Probably Metapneumonic in Origin.

March 22, 1916.

THIS patient, aged twenty-five, came into the hospital with the following complaints: First, slight pain in the left side; second, cough; third, foul-smelling sputum; fourth, profuse sweats.

The Onset and Course.—The patient states that he has had a cough ever since he was a small child. This cough always became much worse in bad weather. He expectorates a great deal at all times, but when the weather is bad the expectoration becomes increased manyfold and becomes extraordinarily foul smelling. The sputum is always purulent, but has never been rusty or blood tinged. Under normal conditions, that is, when the weather is not bad, he will expectorate perhaps a large mouthful in the morning and again expectorates several large mouthfuls during the course of the day. When, as he says, he "catches cold" he will expectorate half a cupful at a time. He thinks that there has been no material change in the cough or expectoration since he was a boy. He has not lost in weight and has an excellent appetite. He works indoors in a dusty wood-working shop.

For about four days before entering the hospital he had a slight pain in the left side and at the same time a chill. He thinks he has had fever. The fever and chills disappeared in a day or two, but he has had profuse sweats.

Previous Illnesses.—The patient has had but few diseases. He does not remember any of the diseases of childhood. He had one attack of acute tonsillitis several years ago and had a tonsillectomy performed for a chronic tonsillitis two years ago. A venereal infection is denied.

Habits.—He uses whisky very moderately, drinks beer occasionally, and is a heavy smoker of cigarettes.

Family History.—The mother is in the Dunning State Hospital for the Insane. The father died when rather young of a stroke of paralysis. He has two brothers and four sisters living and well.

Physical Examination.—The patient is rather pale with dark circles under his eyes, rather sparely built, and of listless expression. The head and neck show nothing especially abnormal except an intensely fetid breath. Even at a distance the odor is almost intolerable. The contour of the chest is rather long and thin; the expansion is slightly diminished on the left side and the entire left chest is flattened to a just perceptible extent. The apex-beat of the heart is in the fifth interspace about the nipple line. It is normal in width and the sounds are all clear. The transverse dulness is not perceptibly increased. The blood-pressure is a little low, 105 systolic, 60 diastolic.

As we examine the *lungs* there are some rather unusual and decidedly interesting findings. The entire right lung is perfectly negative. The borders are all in their normal position and show a normal respiratory excursion. On the left side the respiratory excursion is very much lessened, practically absent. In the lower axillary region on the left side and from the angle of the scapula down there is diminished resonance, and over this area of diminished resonance can be heard harsh breathing and a large number of large, moist râles with a few dry râles interspersed. The noticeable feature is the fact that all the râles are exceedingly large, about the size we are accustomed to hear in the primary bronchi, and a few of them sound almost like tracheal râles.

The abdomen and extremities are negative; the reflexes are all present and normal; there is no general adenopathy.

On admission the leukocyte count was 40,000, with 88 per cent. polymorphonuclears. The urine is negative except for a trace of albumin. The sputum has been examined several times, and I note on the history that one Löffler examination has been made with negative results for tubercle bacilli. An occasional elastic fiber was found.

After being in the hospital only three or four days this patient left because his cough improved greatly. Many previous experi-

ences have taught him that these attacks clear up in the course of a week or two and the cough lessens so that he is able to go back to work. Because of the interest attached to the case I hunted him up and asked him to come back so that I might present him to you.

The range of temperature on the first day of his stay in the hospital was from 100.8° to 101° F. This came down to normal in a day or two. His leukocyte count came back to normal at the same time, and is approximately normal now.

Differential Diagnosis.—When we come to consider the diagnosis in this case the first thing that might enter our minds would be tuberculosis. There are, however, many things which speak against such an assumption. In the first place, the apices, so far as our physical examination goes, seem perfectly normal. Second, the only change of any character which we have been able to demonstrate is in the left lung, low down in the axillary and scapular regions. The fact that the patient has lost no weight, that under ordinary conditions he has no temperature, and that despite the most careful examination of his sputum we are unable to find tubercle bacilli—all these things speak strongly against an old tuberculosis, even of a fibroid type. On the other hand, the patient's condition seems exquisitely dependent upon the weather. In good weather, particularly in the summer, his condition is much better. During wet and changeable weather and especially when, as he says, he "catches cold," the expectoration becomes very profuse and intensely putrid. This has not only happened once or twice, but has happened many times in the course of a number of years, so that it would seem to be a fact established beyond any reasonable doubt.

The next most likely thing would be a chronic bronchitis of a putrid nature. This, in my judgment, can be eliminated because of the fact that we have a very definite focus and this focus exists in only one lung. A putrid bronchitis is in the very nature of the disease essentially bilateral. You will remember that in a recent clinic I made a statement that in my judgment *one of the most valuable signs of bronchiectasis was the fact that we had definite patches of râles which were marked and constant in one posi-*

tion, or at least constant at the time when the patient was showing symptoms. That is the condition which obtains in this patient. While I have not had the opportunity of observing him over a long period of time, I have seen him sufficiently long to know that these large, moist râles are practically constant over the left axillary and scapular regions, and become much more numerous when he is expectorating large quantities.

It seems to me that the only intelligent interpretation of these physical signs, taken in conjunction with the long history of putrid sputum, the absence of any loss of weight, the exacerbations in bad weather, etc., is that we have before us another case of bronchiectasis. The patient's malady dates back, you will remember, to his boyhood, and we cannot ascertain with any certainty whether or not it followed any acute condition, but I think we might hazard a guess upon the subject. My observation leads me to believe that most cases of this sort are *metapneumonic in origin*, and I am inclined to think that this patient had in his boyhood a pneumonia. Now a pneumonia in cases of this sort causes an infiltration of the peribronchial tissue, and especially if resolution be imperfect leaves inflammatory foci which later on contract and distort the bronchi. This is particularly true if there be adhesions to the pleura. It is also highly probable that a persistent bronchitis and infiltration of the bronchial wall itself weakens this to such an extent that the continued coughing produces an ectatic condition.

The fluoroscopic examination has been made and the report states: "A left-sided density is present; this may indicate the presence of a bronchiectatic cavity."

In regard to the size of bronchiectatic cavities there is, I think, a rather erroneous impression that these are apt to be of large size. I think the contrary is true. Most of the cases which I have seen at autopsy have shown cavities varying in size from a filbert to an English walnut. Indeed, in many cases one can hardly speak of a cavity at all in the sense that we have a clean-cut, rounded hole. What one actually finds is a system of bronchi each of which is dilated, sometimes in a saccular manner, sometimes in a more fusiform manner, and all of these together

make up an irregular cavity in which the conditions are exactly right for the retention of fluids and bad for drainage.

With these facts in mind it will become apparent to you why the patient with a bronchiectasis becomes so much worse when any acute infection supervenes. What actually brought this patient into the hospital was an acute exacerbation of his symptoms, and the cause of his temperature is to be sought in the absorption from such an ill-drained area. With rest in bed and ordinary measures of combating an acute respiratory infection the acute symptoms have largely subsided and, as I have already said, he had left the hospital and has only come back because I was anxious to show him to you in the clinic. Let me again impress upon you the fact that many patients with bronchiectasis show few or no symptoms or physical signs except when they acquire a bronchitis in addition.

There is one rather noteworthy feature about this case which illustrates again the great variations in individuals as well as in diseases, and that is, there is no clubbing of the fingers. In the other case recently shown you, in which the process was bilateral and much more severe than this, there was marked clubbing.

Another interesting point is the high leukocyte count (40,000) which this patient had when he came into the hospital. This has returned nearly to normal in the few days which have passed. The explanation for the high count is probably to be found in the increased amount of absorption due to the exacerbation.

Treatment.—The patient is now back at work and has refused to consider operative interference. My own feeling in these cases is that operation should be limited strictly to such cases as are unilateral and where there are in all probability adhesions between the lung and the chest wall. It would, of course, be undertaken only where the symptoms are of sufficient gravity to warrant it. This patient is more annoyed by the foul-smelling sputum than by the coughing. I do not know whether, even if he would consent to operation, much could be accomplished, but it might be possible by draining to obliterate the cavity. In any case, I do not feel warranted in urging such a course upon him,

but merely mention it as something which might prove successful if our medical treatment is unavailing.

There is one thing, however, which I think he should certainly do, and that is, change his occupation. He is working in an exceedingly dusty place, and inhales large quantities of dust every day. This certainly tends to aggravate the condition, and I shall advise him to take up some work which will enable him to spend his time in a purer atmosphere—preferably out of doors.

I think it quite likely that a change of climate, to such a place as Arizona or New Mexico, might be beneficial just as it often is in certain cases of emphysema. An equable, dry, warm climate is ideal. It will not cure the existing lesion, but it minimizes the effects of the weather on the bronchial mucosa, and by preventing bronchitis allows the patient to pass a much more comfortable existence.

RECURRENT THROMBO-ULCERATIVE ENDOCARDITIS

Associated with Marked Jaundice and Extensive Hemorrhages in the Lung and Skin. Pure Culture of Hemolytic Streptococcus Obtained from the Blood. Synopsis of Autopsy Findings.

March 22, 1916.

Present History.—This patient is a Lithuanian laborer, twenty-eight years of age, who came into the hospital on March 13th complaining of swelling of the feet and legs and severe pain in his back. He states that his sickness started six weeks ago with pain and distress over the upper portions of his abdomen, followed by backache. Two weeks later, that is four weeks before admission to the hospital, his feet began to swell. He says the pain in the abdomen was not severe, but has persisted and still makes him very uncomfortable. The backache also is still present and fairly severe. The swelling of the ankles has increased steadily until now he has a rather extensive edema of the lower extremities. In the last few days he has been more short of breath than before.

Previous Illnesses.—With the exception of an occasional headache he knows of no disease which he has ever had except an acute articular rheumatism two years ago. He has never had any recurrence of the rheumatism since that time. He denies a venereal infection.

In his habits he does not differ materially from the ordinary run of laboring men. He drinks a little, smokes considerably, eats and sleeps well—or, rather, did until this sickness began. His bowels and bladder show no abnormalities in their functions.

Family History.—There is nothing of importance in the family history. The mother died of pneumonia at sixty, the father of pneumonia at seventy. The patient has never been married.

Regional Examination.—*Head and Neck.*—The scalp is negative with the exception of a cut which was due to an accident.

The eyes are normal. The teeth and gums show pyorrheal changes. The throat is a little reddened, but not markedly so. The mucous membrane of the mouth is pale.

The Chest.—This is fairly well developed. Expansion is good; the skin white and clear.

The Lungs.—Both lungs show numerous moist râles over the posterior surface, especially at the bases. There is slightly roughened expiration over both apices, but otherwise no abnormality.

The Heart.—The absolute dulness extends from the right edge of the sternum to a point a little outside the nipple line. The upper border is at the third rib. The apex-beat is clearly to be seen and felt in the fifth interspace, 2 cm. outside the nipple line. On auscultation a loud diastolic murmur is heard in the third interspace to the left of the sternum, transmitted over the whole cardiac area. A systolic murmur can also be heard over almost the entire cardiac area. It is louder to the right, but not transmitted to the vessels of the neck. There are no thrills. The second aortic tone is lacking. A well-marked Duroziez sign can be obtained.

The Abdomen.—This is slightly distended and tympanitic excepting over the liver. The liver was thought to be palpable at first, but later examination when the patient became more relaxed disclosed that this was not the case. The spleen is not palpable. The genitalia are negative with the exception of a moderately enlarged prostate.

There is no general adenopathy.

Now please note that these physical signs which I have just read to you were recorded on the 13th, the day of admission; that is, a week ago. We examined the patient carefully the following day and verified them and noted that there was no special change. The diagnosis at that time was an old aortic insufficiency with broken compensation, and there was nothing about the case to stamp it as being in any way different from the ordinary run of such cases. In the absence of a thrill at the aortic area I did not think that there was a stenosis at that orifice. It was noted, however, that the blood-count was rather higher than

we would expect, being 14,000 on the 13th and going up to nearly 18,000 a few days later, with 77 per cent. of polymorphonuclears.

The urine showed a moderate amount of albumin with a specific gravity ranging from 1020 to 1030, and an average of 800 or 900 c.c. in twenty-four hours.

The blood-pressure was 135 systolic, 52 diastolic. The albuminuria was interpreted as symptomatic of the congestion of the kidneys incidental to the broken compensation.

Course and Termination.—On the fifth day of the patient's stay in the hospital he suddenly developed a considerable degree of jaundice. This had not been present in the beginning, as the history explicitly states that the skin was white and clear. The jaundice rapidly became more intense and in a day or two more the skin was a fairly deep yellow. Along with this we noticed that while the degree of decompensation was not very great, the patient was not responding to treatment in the way we felt he should. The heart was becoming more dilated and the patient more dyspneic. Now, whenever an old heart case is not responding to treatment, and especially if there be a leukocytosis present, it should at once arouse the suspicion that there may be recurrent endocarditis.

With this in mind we took a blood-culture and had the satisfaction of finding a *pure culture of the hemolytic streptococcus*. On the 18th, that is, on the same day that the patient developed his jaundice, we also noticed *extensive hemorrhages in the skin of the lumbar region*. On either side was a subcutaneous hemorrhage the size of one's hand, and later on similar hemorrhages developed over the lower extremities.

The temperature, which was normal on admission, had now risen to a very marked degree, reaching 105° F. on the 19th. At the same time the jaundice and hemorrhages developed the patient complained of pain in the side, and examination of the lungs showed an area of *bronchial breathing* posteriorly at the level of the angle of the scapula on the left side, and covering an area the size of a silver dollar. The bronchial breathing was not as clear as we expect to find in pneumonia, and around this small focus was a similar area of markedly impaired resonance. Numer-

ous moist râles and increased breath sounds could be heard, but no true crepitant râles. A number of moist râles could be heard on the right side as well.

The jaundice became deeper and deeper, the consolidation in the left chest became more marked, the patient became more and more dyspneic, and the general picture assumed that of a most acute sepsis. The finding of hemolytic streptococci in the blood made it fairly clear that we had an acute exacerbation of his old endocarditis, and this was our diagnosis.

What has puzzled us so greatly up to the present time is the explanation of the jaundice, the hemorrhages, and the consolidation in the lung. Repeated and careful examinations of the upper abdomen failed to reveal any tenderness over the liver or gall-bladder—not even as much tenderness as one would expect with a venous congestion of the liver due to a broken compensation.

The patient is perfectly conscious at the present time despite the fact that he is desperately ill, and still gives intelligent answers to questions propounded to him by an interpreter. We are entirely unable to find any local explanation for the jaundice—such as a cholangitis or cholecystitis. The possibility of a condition resembling acute yellow atrophy has occurred to us, but the liver is not diminished in size, and examination of the urine, after evaporating considerable quantities, has failed to show leucin or tyrosin crystals. We are driven, then, for lack of any better explanation to conclude that the jaundice is due to the hemolytic streptococcus which was isolated from the blood and that it is a purely septic condition. I do not remember ever having seen a combination of so intense a jaundice and hemorrhages as extensive as these in a septic condition, although I have seen each of them many times separately; that is, I have seen septic conditions with jaundice and septic conditions with hemorrhages. There is, of course, no inherent reason why both may not be entirely due to the sepsis, and that is the conclusion which I have reached.

In a case of endocarditis with a badly dilated right heart, and with dulness suddenly developing in the lung, the natural assumption would be that there was a pulmonary embolus which

came from a thrombus in the right auricle. In this case, however, the pain did not come on with great intensity nor was there any hemorrhage in the beginning, such as we expect to find with pulmonary infarcts. Indeed, the physical findings are at the present moment so extensive that it seems almost incredible that a pulmonary infarct of such magnitude should occur without producing instant death. The alternative would seem to be that an ordinary pneumonia, possibly of hypostatic character, has developed, although this does not have the characteristics of hypostatic pneumonia and is confined to the left side. For the last two days the patient has been expectorating almost pure blood, and this symptom hardly can be harmonized with an assumption of a hypostatic pneumonia.

Another possibility is that small emboli from the right heart produced small infarcts which became infected by the circulating blood, producing a true embolic pneumonia. This seems to me the more likely conception, although the condition is a very obscure one.

To sum up, then, our findings as well as we can, we *interpret this case as one of endocarditis dating back two years and originating, beyond doubt, from the acute articular rheumatism which the patient had at that time. A recurrence of his endocarditis has taken place, as evidenced by the blood-culture, and the jaundice and cutaneous hemorrhages would seem to be the expression of a profound general sepsis.*

The prognosis is, of course, absolutely bad, and it is not likely that the patient will live more than a very few days. If any further changes develop I will report them to you at the next clinic.

Autopsy Findings (March 27th).—The subsequent course of the case I showed you at the last clinic was not unexpected. The patient's septic condition became more and more pronounced and he died a few days after he was shown to you. We were fortunate enough to secure a postmortem examination, which disclosed essentially the following: A recurrent thrombo-ulcerative endocarditis on the aortic valve with both insufficiency and some stenosis. The kidneys showed nothing abnormal except

the scars of old healed infarcts. The liver showed a slight venous congestion, but was not enlarged, and *not a drop of bile could be squeezed from its cut surface.* The gall-bladder showed no abnormalities. The left lung showed an intense hemorrhagic infiltration in its entire lower portion, which at first sight resembled closely a hemorrhagic infarct, but the most painstaking dissection of the vessels failed to show any embolus or thrombus to account for the condition. There was a slight fibroid tuberculosis at each apex which undoubtedly gave rise to the harsh breath sounds at the apices which were noted when he first came into the hospital.

In view of the pathologic findings just noted, and especially in view of the absence of all local conditions in the liver, I think we are justified in assuming that the jaundice was septic, that is, hemolytic. It is frequently asserted that there is no such thing as a pure hematogenous jaundice, but it seems to me that this case certainly speaks in favor of it. It should be remembered that the jaundice developed before the lung condition and before the hemorrhages into the skin.

In regard to the lung, the condition is a little more difficult to interpret. The pathologist who performed the necropsy regards the lung condition as being due to a pure hemorrhage. This would mean that the hemorrhages into the lung tissue are to be regarded in precisely the same light as the hemorrhages into the skin.

A CASE OF INTERMITTENT CLAUDICATION

Circulatory Disturbance in the Lower Limbs. Demonstration of Sclerotic Vessels in the Röntgenogram. Suggestions as to Treatment.

April 19, 1916.

Present History.—This man, a Swede, forty-seven years of age, by occupation a bartender, came into the hospital because of a superficial ulcer of the leg with pain in both calves. The ulcer on the leg has been present for about twenty years and never becomes completely healed. The pain is shooting in character, varies in intensity from day to day, and is always worse in damp weather. He thinks he has lost 14 or 15 pounds in weight during the last year. He was operated upon three or four years ago at the Presbyterian Hospital for varicose veins. He had a gonorrheal infection twenty years ago; he denies having had a chancre.

Habits.—He takes several glasses of beer and several drinks of whisky a day and has done this for years. The bowels are normal; he gets up once or twice at night to urinate.

The *family history* is negative.

Physical Examination.—This patient weighs about 155 pounds and does not appear particularly sick.

Head and Neck.—The pupils react both to light and to accommodation. The teeth and gums are in poor condition; the throat is slightly red. There are no other abnormal findings.

The Chest.—The heart and lungs are negative. The blood-pressure registered 176 systolic, 100 diastolic, at the time when he came into the hospital on March 23d. Since that time the blood-pressure has dropped down to 100 systolic and about 48 diastolic.

The *abdomen* and genitalia are negative.

The *arms* show nothing special except that the brachials pulsate rather markedly and are very sclerotic.

The *legs* above the knees are normal. Below the knees we see a few superficial leg ulcers that do not seem to have any particular bearing on the man's condition.

The *urinalysis* shows a considerable increase in amount, 3000 c.c., specific gravity, 1012, a very slight trace of albumin, no sugar, and a number of casts, mostly finely granular.

The *Wassermann* reaction was negative on both blood and spinal fluid.

The Blood Examination.—The white count shows 10,800; the differential count gave 53 per cent. polymorphonuclears, 5 per cent. large mononuclears, 37 per cent. small mononuclears, 3 per cent. eosinophils, 1 per cent. basophils, 1 per cent. transitionals.

Diagnosis.—This man comes into the hospital complaining of pain in the legs which has been present for a considerable time. As a rule it is not bad when he is lying down and often disappears. The superficial leg ulcers are entirely inadequate to account for the pain. When a man of middle age complains of pain in the legs the first thing to consider in the diagnosis is the possibility of tabes. But there is nothing to support that assumption. The Wassermann reaction on both blood and spinal fluid is negative. There is no history of syphilis; the knee-jerks are present, the pupils react normally, and there are no other findings upon which to base a diagnosis of tabes. The spinal fluid was clear and colorless, the Nonne and Noguchi tests were perfectly negative, and there were only two cells to the cubic millimeter.

The next thing to think of is a neuritis, particularly an alcoholic neuritis. This man has been a hard drinker. But in a neuritis there is tenderness of the muscles, and when we pick up the muscles of the calf of the leg we find absolutely no tenderness. In a case of such long standing as this one there would probably be pains elsewhere, either in the arms or some other part of the body.

We next find upon inquiry that he rarely ever gets these pains when he is lying down or when he is sitting up, and then they are rather feelings of numbness than actual pain, but if he starts to walk or goes up and down stairs the severe pains soon appear. After he walks a little while he has to limp. Sometimes the pains come on after walking half a block, or even up and down

a long ward in the hospital, and at other times they develop almost immediately. (Has patient walk back and forth across amphitheater.) He has walked now not to exceed fifty yards and he says the pain is coming on. You can also notice that he has commenced to limp. Does that suggest anything to you?

VISITOR: Intermittent claudication.

DR. WILLIAMSON: It is a typical case of intermittent claudication. The superficial leg ulcers are insignificant and could hardly cause pain of any moment. We can do several other things that will substantiate the diagnosis. So far as the circulation is concerned, the upper portions of his legs are comfortably warm, but as we go down *toward the feet we find them as cold as if they had been on ice and very markedly blue*. On investigating the dorsalis pedis and the posterior tibial arteries, it is impossible to feel the pulsation in the dorsalis pedis on the left side. On the right side there is a very feeble pulsation. On superficial palpation you might find no pulsation at all. When I produce a pallor of the skin by pressure of my finger, it is several minutes before the pallor disappears, showing the greatly retarded circulation.

If we are correct in our assumption that this is an intermittent claudication and that the dorsalis pedis is hardly pulsating at all, we ought to be able to see that in the Röntgenogram. We have had *x*-ray plates of the legs taken (exhibiting plates in shadow-box) and we can see the dorsalis pedis distinctly sclerotic and the posterior tibial markedly sclerotic. In view of the fact that there is no tenderness anywhere, that he has this striking clamminess and coldness of both feet from about the lower third down, and in view of the fact that the reflexes are both present and normal, we can rule out tabes and alcoholic neuritis with certainty. Osteomyelitis could hardly be considered with such a picture as this.

Now I shall say a few words about the subject of intermittent claudication. The best name that I know for it is that originally given it by Erb, who spoke of it as *Dysbasia angiosclerotica*. The pathologic basis lies in the sclerosed blood-vessels. As a result of the deficient supply of blood these patients suffer with cold and clammy feet; the pain is not very pronounced as long as they are quiet, but as soon as they do anything that demands an

extra supply of blood to the lower limbs the pains increase. The pains are sometimes so severe that the patients have to sit down. They rest for a few minutes and then can walk again.

Fig. 33.—Intermittent claudication showing marked sclerosis of the artery.

I believe that this case has been wrongly interpreted by us up to this time because he had these varicose ulcers, which have no

particular significance. The diagnosis is confirmed by the arterial findings and by the *x*-ray pictures.

These cases are frequently diagnosed as rheumatism. You must bear in mind that the patients always experience the most pain when a greater blood-supply is required to the muscles. In this man's case the pain is particularly severe when he walks up and down stairs because it produces a greater effort on the part of the arteries, and in consequence of that, a greater amount of pain.

Prognosis and Treatment.—We can do little or nothing for the sclerotic arteries. On the other hand, we can do quite a little for these patients if they can be made to live a proper life. They should avoid all kinds of muscular effort and should keep the feet elevated at least part of the time. Bier's hyperemia has been suggested and is said to have helped in some cases, but I am a little doubtful of its value. Potassium iodid has been used in these cases and in some instances it has apparently been of some benefit.

We discussed the possibility of syphilis in this case because often such vessel changes are due to a syphilitic arteritis. But with his negative history, the negative Wassermann on both blood and spinal fluid, and the negative cell count we have no reason for considering the vascular changes as syphilitic in origin.

Another treatment that has been suggested is the giving of various combinations of alkaline salts. In Europe a mixture known as antisclerosin has been used very widely, the belief being that by the use of different salts in solution the deposits in the blood-vessels could be favorably influenced. Personally, I do not think that they have any influence on such cases. We must remember that many of these cases come on from overwork in these muscles. I have a patient in private practice who has a very high-grade sclerosis of the posterior and anterior tibials and a typical intermittent claudication. He formerly walked long distances while on deer-hunting and fishing expeditions, but during the last couple of years he has had to give up this sort of thing.

This is only a moderately developed case; he has a little discomfort all the time, but it gets very much more marked when he walks any distance.

A CASE OF CEREBROSPINAL MENINGITIS

Demonstration of Meningococcus in Cerebrospinal Fluid. Recovery after Two Injections of Flexner's Serum.

April 19, 1916.

THIS patient, an American, twenty-four years of age, by occupation a machinist, came into the hospital on April 13th, complaining of headache, backache, and stiffness of the neck. The illness started two days before he came in, when he awakened in the morning feeling sick and vomited three or four times. A headache developed which became worse, and on the following day he had a severe chill lasting two hours. The backache came on the morning he entered the hospital. He had a slight cough and expectorated a little yellow sputum. He says he has had a cold in the head and nose all the time. The vomiting has continued, occurring about once an hour. So far as he knows the vomiting was really the first symptom and has persisted since the onset. There has been no blood and not very much food in the vomitus. The backache was confined to the lumbar region and was very sharp. The headache was frontal in the beginning, then went to the back of the head, and then down to the nape of the neck. He says that the chill was very severe—he shook for two hours. The afternoon he came into the hospital he had another chill, but this was of shorter duration. He has had a cough for about a month, but thinks this had nothing to do with his present condition. The bowels have been constipated. There have been no night-sweats, no loss of weight, no swelling of the feet and ankles, and no intestinal or urinary disturbance.

Past Illnesses.—He had measles, chicken-pox, and tonsillitis in childhood. He had gonorrhea three years ago; he denies having had a chancre.

Family History.—His mother is living and well. His father died of a cerebral hemorrhage at seventy. His brothers and sisters are all living and well. He has never been married.

The Temperature.—On admission the temperature was 101° F., pulse 88, respirations 22. This was at 10 o'clock in the evening. The next morning the temperature was 101.4° F., which was the highest it reached that day. Yesterday it was 99.2° F.—a moderate degree of temperature.

Physical Examination.—On admission the patient appeared acutely ill, was restless, somnolent, and did not answer questions well. He rested mostly on his side with the knees drawn up, but was able to lie on his back. He complained of pain in the back of his neck. Shortly after entrance he vomited 6 ounces of stomach contents which showed no blood.

Head and Neck.—The scalp was negative. The pupils reacted well to light and accommodation. The throat was red, the tongue dry and furred. The teeth and gums were in fair condition. The neck was bent distinctly backward and one could lift him up by slipping the hand under the back of his head. He could move his neck sidewise, but the muscles were definitely rigid.

The *chest* showed nothing abnormal, either as to heart or lungs.

The Abdomen.—Examination revealed no abnormal findings; the spleen was not enlarged. The genitalia were negative.

Extremities.—There were one or two scars on the anterior surface of the tibia; no lesions were present on the skin. There was no edema.

DR. WILLIAMSON: What is the diagnosis?

VISITOR: Probably a meningitis following an attack of grip, but it might be a tubercular meningitis following a lung condition.

DR. WILLIAMSON: It is suggested that it might be a meningitis. I will read you the results of the laboratory work, and then we will see. The urine was normal in quantity, with a specific gravity of 1020, a mere trace of albumin, no sugar, an occasional

white blood-cell, no casts, and an acid reaction. The blood-count showed 30,400 white cells and the great majority of these were polymorphonuclears.

We then did a spinal puncture and removed 30 c.c. of opaque fluid with *a cell count of 25,000 per cubic millimeter*. The fluid was under increased pressure. The differential count showed 96 per cent. polymorphonuclears, 4 per cent. lymphocytes. In addition to this, smears showed a Gram-negative intracellular diplococcus which was biscuit shaped. Now what is the diagnosis?

VISITOR: It sounds like a cerebrospinal meningitis.

DR. WILLIAMSON: It is a perfectly classical and typical case of cerebrospinal meningitis. There are two or three things about it that are interesting. The onset with vomiting does not throw much light on the subject, for that occurs in all sorts of diseases. When a young man who has had a little cough begins to vomit in the morning, cerebrospinal meningitis is not the first thought. Then he developed a frontal headache which found its way into the occipital region, and then into the cervical region. You will remember that in epidemic cerebrospinal meningitis, of which this is a typical case, the backache is due to involvement of the meninges over the spine. There is no age limit to this disease; it may occur at any age.

A number of other symptoms were present. To begin with, he had a marked hyperesthesia over the legs that has almost disappeared now. He had a well-marked *Kernig sign* and that is still present. The reflexes when he came in were tested very carefully. There was no Babinski on either side; the Achilles tendon reflex was present, the patellar reflex was present and increased, the Kernig was present on both sides, the abdominal and cremasteric were both present—nothing remarkable about the reflexes except that the knee-jerks were increased and there was a well-marked Kernig on both sides. The backache was very severe, especially in the lumbar portion.

The first point of interest about the case is, therefore, the mode of onset. The vomiting and headache over the front of the head would hardly make one think of cerebrospinal meningitis

the first day. The second point of interest is the certainty with which the diagnosis can be made. The large amount of fluid obtained by lumbar puncture, the increased pressure, the opacity of the fluid, the high cell count, the enormous preponderance of polymorphonuclears—96 per cent. in this case—and then the finding of the *meningococcus of Weichselbaum*—all these make the diagnosis perfectly certain.

It was suggested that this might be a case of *influenzal meningitis*, but we must remember that the meningococcus has been found in the nasal secretion just about as frequently as the influenza bacillus. Does the organism find its way to the brain through the lymphatics, or in a more roundabout way? The probability is that the organism gets into the blood and that it has a special predilection for the meninges. Although the most direct route is through the lymphatics, I believe that in all probability it reaches the meninges through the blood-stream. It may be that both forms of distribution occur.

How are these cases carried? What are the dangers associated with them? They are probably carried through the nasal secretions, and it has been noticed for many years that the communicability is greatest where people are herded together in damp dwellings, under poor hygienic conditions. In the well-kept hospitals where the secretions are carefully destroyed there is very little danger of spreading the disease. This is particularly true where the physician recognizes what the condition is. How does it happen that other patients sometimes develop the trouble in spite of isolation?

VISITOR: The germ is probably present in a good many nasal secretions.

DR. WILLIAMSON: The probability is that this patient expectorates enough of the meningococci so that the people around him also harbor them in their nasal secretions, although they may never develop the disease. Many carry these germs around and under exposure to cold the disease may develop. The eruption in these cases has been looked upon as characteristic, but my experience is that, as in typhoid fever, one cannot rely exclusively on it for diagnosis.

Treatment.—This case has responded excellently to treatment. The mortality in such cases used to be high, varying from 20 to 80 per cent., on the average 40 to 50 per cent. The treatment has been greatly improved since we have had the *Flexner serum*, and the mortality has been reduced to about 20 per cent. The proper thing to do in these cases is to put the patient to bed, make a lumbar puncture, and, if one finds the diplococcus of Weichselbaum, the serum should be immediately introduced; 30 or 40 c.c. of fluid should be taken out and replaced with the serum. It is often good practice to remove as much as possible. This patient had 30 c.c. of serum on each of two succeeding days. He feels pretty well now with the exception of the marked stiffness of the neck. The quick result I attribute to the fact that we saw the patient early in the disease, made the diagnosis promptly, and administered the serum at once.

Conclusions should not be drawn from one case. We find some cases so severe that they die in a few days, others that go along for two or three weeks—these are the average cases—and still others that last six or seven weeks, and even longer. I do not believe this man is getting well because it was a mild case, for we have evidence from the enormous cell count that it was a severe infection, but I do think that the good effect is entirely due to the early use of the serum. He has had no other treatment and we shall give him nothing else unless he shows symptoms of getting worse.

A word or two about the meningococcus in the clinical sense. It is an organism which does not show very great resistance. It is said not to resist the effect of germicides or antiseptics as do some other organisms, which is very fortunate, for it thereby enables us to get such good results. This might have been a pneumococcus infection, which might occur even without a pulmonary involvement. The question is asked whether one should use serum in a case of pneumococcus infection. I think it a good plan to have the serum ready, and if the spinal fluid comes out strongly turbid the serum should be injected, irrespective of what the bacteriology may be. If it does not prove to be a meningococcus then it is not necessary to give any more serum.

In severe cases as much as 45 c.c. of serum should be given and repeated, if necessary, in twelve hours. In average cases it is customary to inject 30 c.c. on each of four successive days. In this case the response to treatment was so brilliant that he received only two injections. Within forty-eight hours his condition was nearly normal except for a little cervical rigidity and a fraction of a degree of fever.

NOTE.—May 12, 1916. This patient made an extraordinarily rapid and uneventful recovery.

CLINIC OF DR. JAMES T. CASE

X-RAY DEPARTMENT, ST. LUKE'S HOSPITAL, CHICAGO

THE PRINCIPLES OF FLUOROSCOPY OF THE STOMACH

Description of Modern Fluoroscopic Apparatus. Relative Value of Plate and Screen Methods in the Study of the Alimentary Tract.

Detailed Technic in Examination of Esophagus, Stomach, and Duodenum. Fluoroscopic Palpation only Certain Method of Detecting Adhesions.

Normal Indentations of the Barium-filled Stomach. Impossibility of Differentiating an Inflammatory Mass and an Early Malignancy from a Defect in the Gastric Shadow.

THE gentlemen will please come into the fluoroscopic room. Our *x*-ray work is divided up into that which is essentially fluoroscopic, that which is roentgenographic, or by means of plates, and that which is therapeutic. Today we will discuss the general principles of fluoroscopy, and the roentgen findings in relation to the normal esophagus and the normal stomach and duodenum.

The nurse will shut the doors carefully so that the room will be thrown into total darkness. It is quite useless to attempt any fluoroscopic work until the eyes have become accustomed to the darkness by a stay of at least fifteen minutes in the dark room. Let me emphasize that this preparation is absolutely essential, for the luminous sensibility of the eyes of the observer plays an important part in fluoroscopic work, and it is necessary to acquire a high degree of retinal sensibility to the fluorescence of the screen in order to make reliable observations. When one does his work at dusk or at night the sensibility is, of course, great, without any preparation. In the daytime a period of preparation in absolute darkness should be planned for.

Professor Béclère, of Paris, who is considered the dean of French roentgenologists, made an extensive study of this subject as long ago as 1899. His observations, published in the *Archives d'Electricité medicale*, No. 82, October 15, 1899, proved by precise measurement that after ten minutes in the dark the sensibility of the retina to the light of the fluorescent screen has become 50 to 100 times greater than it was on emerging from broad daylight, about 200 times greater after twenty minutes' observation, and still more increased after a longer interval. Very often we have a visiting doctor remark, after having remained in the fluoroscopic room for an hour or more, that he thinks he can see very much better than when he first came in. Sometimes the patients express a certain amount of disturbance at the delay of fifteen minutes that this preparation of the eyes entails, so it is necessary to explain to them that in reality we are saving time, not wasting it, by getting our eyes accustomed to the darkness.

When we first commenced fluoroscopic work in this hospital it often happened that the attending physician would come directly from the well-lighted wards to the fluoroscopic room, and ask to see a certain case, and then when the *x*-ray tube was turned on express considerable disappointment at not being able to see satisfactorily. It has been said that one makes more mistakes by not looking than by not knowing, but even to look requires a certain preparation.

Modern fluoroscopy requires that the work be done in a thoroughly darkened room. All cracks, even the very keyholes, should be covered and made impervious to light. Overhead wires must be so installed that there will be no "corona" or glow upon the wires during the passage of a high-tension current.

A fluorescent screen is an ordinary sheet of cardboard, containing on one side a coating of some chemical (barium platinocyanid or calcium tungstate) which fluoresces on exposure to the *x*-ray. In the earlier days of roentgenology it was more convenient to have this screen form the end of a dark pyramidal-shaped box, light-tight except for the eye-holes, which enabled the use of the screen without darkening the room. A sheet of

lead glass over the screen protects the screen from deterioration and the observer's eyes from the roentgen rays. On account of the necessity of preparing the eyes, manipulating the patient, and properly protecting the patient and operator from the ray these pyramidal boxes have been practically discarded, and no up-to-date physician would attempt to do fluoroscopic work with one of them.

QUESTION.—Much has been said concerning the danger of fluoroscopy. How much real danger is there in fluoroscopic work at the present time?

DR. CASE: Under the old conditions of protection or, rather, lack of protection from the *x*-rays, fluoroscopic work was exceedingly dangerous, not to the patients, but to the observer, although occasionally even patients were injured. Lead glass was unknown. The small box fluoroscope, such as was mentioned a few moments ago, was in common use. Diaphragms and lead-glass bowls for cutting off all but the useful cone of *x*-rays were not yet invented. In fact, it was customary in the early days for the roentgenologist to test the hardness of his tubes by examining his own hand with the box fluoroscope. No one at that time appreciated the danger of this method, although its practice led to very many severely burned hands in the earlier *x*-ray workers.

The modern types of fluoroscopic apparatus (Figs. 34 and 35) insure thorough protection from the *x*-ray. The tube is enclosed in a protective lead box from which no rays emerge except from the opening in the diaphragm. The size of this opening can be adjusted at will from the front of the instrument, and it is so arranged that at all times the cone of emergent rays must impinge upon the fluorescent screen in front of the patient. This screen is backed with heavy lead glass. You will notice that we employ here two thicknesses of the heaviest lead glass obtainable. When tubes of ordinary penetration are employed practically no rays emerge into the room. I say practically, because speaking from a strictly scientific standpoint, it is impossible to cut off all the ultra-hard rays, even with the best protective material. The danger from these, however, is probably negligible. We consider the operation of a properly built fluoroscope at the present time no

more dangerous than scores of other occupations in which men are daily engaged without any thought of peril. I assure you if I thought the *x*-rays would injure me I would promptly give up the work. On account of the patient's relatively short stay in the

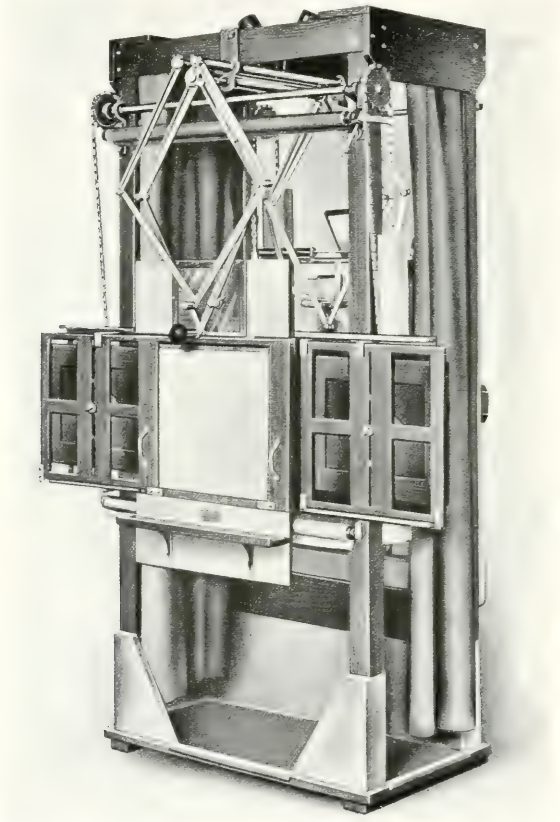


Fig. 34.—A satisfactory instrument for upright fluoroscopy.

fluoroscope, even in the very prolonged examinations, it is hardly necessary to take the trouble to interpose any protective material between the tube and the patient. Nevertheless, as a precaution against the occasional case of special susceptibility to the *x*-rays, a sheet of aluminum one millimeter in thickness placed between

the tube and the patient will filter out the soft rays which in overdose produce dermatitis. A piece of sole leather serves about as well as a millimeter of aluminum. In all of my work I have known no patient to show a reaction from the *x*-ray during the diagnostic work. Of course, in therapeutic work a reaction is often seen. Most often it is deliberately produced.

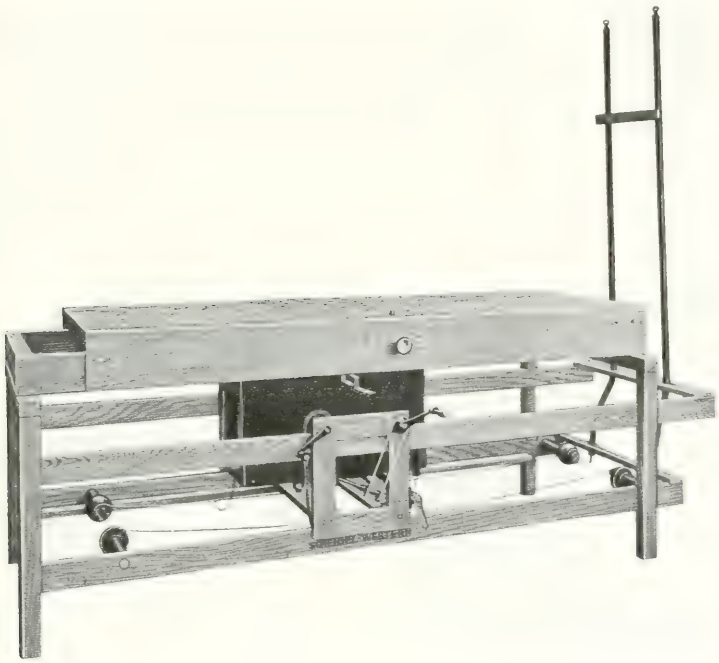


Fig. 35.—A satisfactory instrument for horizontal fluoroscopy. There are many satisfactory types of these instruments on the market.

The question of idiosyncrasies, of course, arises. There are some of us who believe that in occasional very rare instances one encounters a patient who is unusually susceptible to the *x*-ray. I formerly agreed with the majority, who hold that there is no such thing as *x*-ray idiosyncrasy, but within the last three or four years I have had 2 cases that have convinced me that such hypersusceptibility does exist, although, as I said before, very rarely.

The most dangerous and yet the most valuable part of fluoroscopy is palpation underneath the fluorescent screen, without which the fluoroscopic image would be of very little value. The roentgenologist must place his hand (or some palpating instrument in place of the hand) between the patient and the screen in order to manipulate the abdomen, and observe under the screen the effect of this manipulation. As soon as we begin our work you will see how easy it is to manipulate the stomach and duodenum, the cecum or other parts of the colon, to lift up the low transverse colon or to separate the two legs of the hepatic loop, and otherwise test the mobility of the colon. This is the only method by which definite conclusions can be reached as to the presence or absence of binding adhesions. In the stomach, for instance, an unusually transverse position suggests adhesions involving the pylorus and the first part of the duodenum (hepatofixation), but no one would dare to make a diagnosis of pericholecystic adhesions from this *appearance* alone. Satisfactory palpation must be practised. One must determine by actual palpation under the screen that it is impossible to pull the pyloric end of the stomach away from its right-sided position under the liver, before concluding that such binding adhesions do exist. In very heavy individuals, especially those tending to obesity, proper manipulation to determine this question of adhesions is often impossible, and then the question must be left unanswered. Without intelligent palpation carried on under fluoroscopic guidance you can hardly hope to successfully detect binding adhesions of the cecum, appendix, terminal ileum, or any other portion of the bowel.

Much has been written and said concerning the relative value of the plate and screen methods. Those fully acquainted with the details of technic of European roentgenologists realize that *x*-ray work, as practised on the Continent, especially in relation to internal medicine, is essentially fluoroscopic. The making of plates and prints is a matter of very minor consequence in the average European roentgen examination department. In America, on the contrary, the examinations have, from the first, been roentgenographic rather than roentgenoscopic. It is true that

pioneer work in fluoroscopy was done in this country, Dr. Francis H. Williams, of Boston, having practised fluoroscopy of the chest in the earliest days of roentgenology; but the sad experiences of many early pioneers in fluoroscopic work gave justly a decided setback to the development of the screen methods until such time as proper protection could be afforded. Nowadays such properly protected instruments are available.

In the study of fractures and in the recognition and localization of calculi and foreign bodies, roentgenography is not only the more reliable, but it also affords the necessary permanent records of the case. In the study of diseases of the thorax the plate records are desirable, and in the study of pulmonary tuberculosis they are absolutely necessary for interpretation and comparison with other clinical findings.

In the study of the alimentary tract, however, fluoroscopy is the method of choice or, rather, we may say, the method which gives us by far the greater yield of valuable information concerning the case. The roentgenogram furnishes a representation of static conditions, the shape (at the moment) and the position; but, of far greater importance in the majority of cases, roentgenoscopy furnishes data concerning mobility, motility, coincidence of pressure pain points with filling defects, spastic manifestations in the stomach and other questions relating to function, which, by roentgenograms alone, either cannot be shown at all, or only with difficulty and at considerable expense. After a large experience in roentgenographic and roentgenoscopic examinations of the alimentary tract, I can truly say that with the exception of gall-stone and duodenal ulcer cases, in comparatively few instances have the roentgenograms added any material fact of importance to the fluoroscopic findings.

In the average patient fluoroscopy of the stomach is quite satisfactory, no plates being needed except for the recognition of possible gall-stones. Gall-stones can be shown by the *x*-ray method in half the positive cases, but in patients too heavy for favorable fluoroscopy, one may have to depend entirely upon serial roentgenograms. Of course, when making palpation under the fluorescent screen, the hand must be protected. Palpation

with the bare hand cannot be too strongly condemned. One occasionally finds a fluoroscopist who will content himself with a leather glove, but emphasis must be laid upon the necessity of using heavily leaded gloves. Of course, the ray which reaches the examiner's hand is filtered through the aluminum and leather which should always be interposed between the tube and patient, through the leather, aluminum or whatever material the table-top or back is composed, and through the body of the patient and the material of the glove. The experience of the last fifteen years shows that lead gloves are practically safe, but for added security a mechanical palpator may be employed. This mechanical palpator is made of wood or aluminum, and is spoon-shaped, built in such a manner that the bowl of the spoon may be placed underneath the screen, while the handle is on the upper side, where the holding hand may be further protected by the lead glass which backs the fluorescent screen. Only those who have experienced the satisfaction of palpating the barium-filled stomach or bowel under the fluorescent screen can appreciate the value of visualized palpation under fluoroscopic guidance.

Now that we have been in the dark room for fifteen minutes you have observed that we are not in total darkness, but that there is a subdued light in the room, which comes from a small, inverted light on the ceiling. The diffuse light in the room permits one to read large type and make occasional notations of our findings, yet it is not bright enough to interfere with the preparation of our eyes for fluoroscopic work. This faint light is connected with the *x*-ray foot-switch, so that when the *x*-ray is turned on the subdued light is automatically turned off, and, vice versa, when the ray is turned off the light comes on again.

You will observe that we have two fluoroscopes, one for upright and one for horizontal work. There are now on the market instruments which combine these two in one for the convenience of the physician whose office space is limited and whose *x*-ray work is not extensive. We make it an absolute rule to examine every patient, when possible, in both the upright and the horizontal positions.

QUESTION: If only one instrument were available, which would you recommend, the horizontal or the upright fluoroscope?

DR. CASE: I do not see how one can do adequate work in fluoroscopy without being able to examine patients in both positions. If one of the instruments is dispensed with, the work will be correspondingly less efficient. In chest work the upright position is preferable, although it is occasionally desirable to examine the chest of a patient who is too sick to sit or stand. For esophageal work the upright position is essential, although occasionally certain lesions may be shown more clearly with the patient horizontal. In gastro-enterologic work about nine-tenths of the information required is secured from the examination in the horizontal position, but the remaining information, which must be obtained in the upright position, is very valuable and cannot be dispensed with.

I have asked only one patient to come in with us today, intending to use her as a text, as well as to examine her. The patient will now stand in the upright fluoroscope. As I sit here before the instrument the patient faces me, hips and shoulders both touching the screen. We see that both lung fields are uniformly and satisfactorily illuminated. The heart and aorta are normal in size and shape. The pulsations of the heart are normal. The excursions of the diaphragm are equal on the two sides. Both the costophrenic and the cardiophrenic angles are normal. The brightness of the illumination of the apices increases as the patient coughs. Will the patient now please be seated for a moment?

This patient has been sent in for examination of the stomach for suspected duodenal ulcer. At the present moment I do not care to know anything more with reference to the clinical findings in the case, lest I lay myself liable to the criticism of having been prejudiced by these findings. It is probably impossible for anyone to avoid being influenced, at least in a small way, by opinions which have been expressed by others. Therefore I only wish to know enough about the clinical suspicion in a given case to enable me to plan to make the examination most effective.

I examine the chest of every patient who comes for fluoroscopic diagnosis. One cannot see too many normal cases, and occasion-

ally we are able to discover unsuspected lesions. I do not pretend, from fluoroscopic study alone, to give an opinion as to the presence or absence of an early pulmonary involvement in any case, although I am sure a gross involvement can easily be seen with the fluoroscope. Fluoroscopy of the chest is of greatest value in the study of the heart and mediastinum, and in observing the costophrenic angles, the excursion of the diaphragm, and the lighting of the apices during coughing or forced respiration. Effusions, even when small, can be easily seen, but for an adequate description of the lungs, especially when an active tuberculosis is suspected, stereoscopic plates of the chest are necessary. In the hands of a competent roentgenologist, who has had sufficient experience in the study of the lungs, stereoscopic chest plates furnish earlier information than can be obtained by the great majority of diagnosticians from physical examination alone. A few very specially trained physical diagnosticians can probably get along without an *x*-ray examination of the lungs, but I know several very expert men of this class who are very reluctant to give an opinion regarding the lungs without the confirmation and check afforded by a stereoscopic chest examination interpreted by a competent man.

QUESTION: Can the *x*-ray demonstrate cavities in the apices?

DR. CASE: In the hands of a competent man *x*-ray examination of the chest by means of stereoscopic plates will reveal any cavity which can be demonstrated postmortem.

QUESTION: Professor —— told us the other day he had recently seen a case of abscess of the lung which the *x*-ray entirely failed to show. Can you explain this?

DR. CASE: I am quite willing to believe that the plates or the *x*-ray examination in that particular case failed to show the evidences which the examiner could interpret as lung abscess, but I am perfectly sure that under perfect conditions of technic, employing stereoscopic plates, the abscess could have been shown.

I will utilize this opportunity to take exception to the statement we so frequently encounter that "the *x*-ray failed." This is a rather trite expression, which I sometimes feel is used to keep the *x*-ray man duly humbled. The truth of the matter, it seems to

me, is not that the *x*-ray failed, but that the man who attempted to do the *x*-ray work failed. The chief factor in estimating the value of the *x*-ray method is the personal factor of the *x*-ray man himself, the man behind the gun, and it goes without saying that the operator in charge of the *x*-ray work must be a physician with a practical knowledge of the clinical aspects of the conditions he attempts to interpret.

Will the patient please stand again? We notice below the left diaphragm a rounded bright area, which we recognize at once as air or gas in the stomach. This is called, by the Germans, the *Magenblase*, or gastric air bubble. We frequently encounter in American literature the German term *Magenblase*. The patient is now handed a glass one-third full of water into which has been stirred a level teaspoonful of barium sulphate.[1] While the patient swallows we observe the passage of the opaque mixture down the esophagus, into the *magenblase*. Entering this, it glides along the inner surface of its wall until it reaches the lower, funnel-shaped end. Here it stays a moment, being slightly lifted up and down by respiration. With every inspiration the gas bubble becomes a little longer and narrower, stretching its lower end until suddenly the barium-water mixture assumes a form of column, reaching from the *magenblase* to the lowest point of the stomach. This much is normal. It is, of course, important to make these tests on the fasting stomach.

When the gastric tone is diminished the barium-water mixture does not form this narrow vertical column, but passes directly to the bottom of the stomach, where it forms a pool with a level upper border and a curved lower border. Another glass is now handed the patient, containing a mixture of cornmeal pap, malted milk, buttermilk, or some other similar thick preparation containing from 1 ounce to $1\frac{1}{2}$ ounces of barium sulphate. A warm food is often very grateful to the patient. The vehicle should be thick enough to retain the barium in suspension for a long time. The patient is asked to swallow this mixture as rapidly as possible, the haste being advisable in order that we may test

[1] Special brands of barium sulphate are on the market which are guaranteed safe for *x*-ray work.

the cardia. Occasionally by accident one observes a certain degree of cardiospasm. As the ingestion of this portion of the opaque meal proceeds, its entry into the stomach is also carefully watched. The narrow vertical column formed by the barium-water mixture gradually widens, and its lower pole drops, as you see, only a couple of centimeters. This stomach seems to have normal tone. In conditions of gastric hypotonicity the second glassful may depress the lower border several inches, the stomach assuming an entirely different shape than that we see here.

We now turn the patient obliquely, so that the right chest is against the screen, the tube being behind the left shoulder. The rays thus pass through the patient obliquely, so that the mediastinal space is clear. You will see that for the entire distance from the clavicles to the diaphragm there is a clear zone between the shadow of the spine and that of the mediastinum. The patient, still in the oblique position, is now asked to drink from the third glass, which, in our practice, contains 7 ounces of buttermilk and 1 ounce of barium sulphate well stirred. During the ingestion of this glassful we especially study the esophagus. The esophagus cannot be recognized directly without barium; even very large tumors of the esophagus are not recognizable on the screen without barium. In the normal esophagus opaque fluids rapidly pass down into the stomach, although physical conditions may occasionally cause the delay of a few seconds at the cardiac orifice. Unless there is a perceptible dilatation of the esophagus above the cardiac orifice one hesitates to pronounce a slight delay as being due to cardiospasm. Although fluids pass rapidly, pastes are propelled more slowly in the shape of cylinders, about the length of a finger. A slight delay higher up in the esophagus with slight regurgitation is not pathologic, but rather the rule. Such delays are often seen at the level of the aortic arch and at the level of the diaphragm. Even in the incipient stages of organic stenosis of the esophagus there is a perceptible delay in the passage of the thicker *x*-ray material, although opaque fluids may pass without difficulty. As a rule, there is a dilatation above a stenosis.

In future hours we will discuss in further detail the examination of the chest and the examination of the esophagus, so more will not be said on this point now.

We see that in this case all the opaque material passes into the stomach without difficulty. The stomach now contains about 18 ounces of a fluid meal. Its lower border is about 3 inches below the navel, but in my opinion this is no lower than we ordinarily encounter it in the average symptomless woman.

The shadow of the normal stomach, barium-filled, is a characteristically complete shadow, subject to certain normal indentations. These normal indentations are as follows:

(*a*) The splenic notch usually present at the upper border of the greater curvature, due to the pressure of the spleen against the greater curvature. One may judge thereby as to the size of the spleen.

(*b*) The changes in shape of the stomach shadow produced by the peristaltic waves are varying but characteristic, and are easily recognized under the fluoroscopic screen or by a series of roentgenograms.

(*c*) The pyloric sulcus, the break between the shadow of the stomach and the shadow of the first portion of the duodenum (variously termed bulbus duodeni, duodenal bulb, stomach cap), normally about 1 cm. in width.

Excluding these normal indentations, any defect in the gastric shadow must be regarded as a mass, either extra- or intraventricular, and the identity of the mass causing this defective filling must be determined. It is probable that we should regard every filling defect as suspicious of malignancy, but it is practically impossible in the very early stages to differentiate from the *x*-ray findings alone between an inflammatory mass, as, for instance, a callous ulcer and an early carcinoma. Even the surgeon at operation may find it impossible to make this differentiation, the question being settled finally only by the microscope. Some authors hold that all tumors, including carcinoma, adenoma, myoma, fibroma, sarcoma, and indurated ulcers, should be regarded clinically as gastric carcinoma, because they can be differentiated one from the other only by careful microscopic examination of

the specimen after its removal. There are cases, particularly the early ones, where from the roentgen examination alone one may only say there is a mass, without settling the opinion whether it is due to ulcer with inflammatory induration or to malignancy.

Let us now watch the peristaltic waves. With the patient erect, you see they begin at a point just above the middle of the stomach, proceeding slowly to the pylorus, fifteen or twenty seconds being required to make the journey from the point of

Fig. 36.—A composite tracing by Rieder made from twelve consecutive roentgenograms of a normal stomach. These were made within twenty seconds, the patient holding the breath meanwhile.

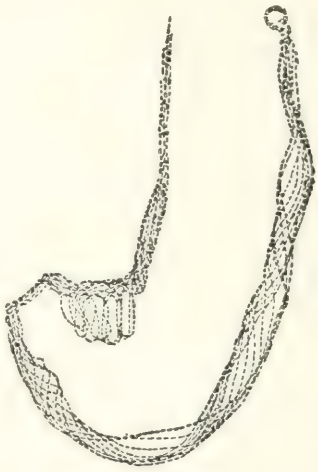


Fig. 37.—Another composite tracing by Rieder made in a manner similar to Fig. 36 in a case of carcinoma of the stomach.

origin to their disappearance into the pyloric ring. The peristaltic waves in this case are normal. The depth of the waves increases as the waves near the pylorus, until in the prepyloric region the incisures are very definitely seen, forming the so-called antrum. It was formerly thought that the pyloric antrum was an anatomic entity, but we know from our roentgenologic studies that the antrum is ever changing, owing to the progress of the peristaltic wave. You see here that each antrum, as it is formed,

is squeezed pylorusward, becoming smaller and smaller until it disappears, by which time a new antrum has formed.

This is a so-called three-wave or three-cycle stomach. It is possible to make tracings of the outline of the stomach from consecutive instantaneous roentgenograms, which will illustrate, in a very graphic manner, the progression of the peristaltic waves. When we turn on the light I will show you a composite tracing

Fig. 38.—The normal stomach, patient prone.

made from twelve consecutive roentgenograms, which were produced within twenty seconds, first in a normal case (Fig. 36) and then in a case of carcinoma of the pylorus (Fig. 37). The tracing of the carcinoma case will illustrate the inelasticity of the pars pylorica, which was the seat of the tumor.

Surmounting the stomach we find the duodenal bulb, or first part of the duodenum (Fig. 38). This is a cone-shaped shadow, which has been likened, by Cole, to a cap, although this term has

not yet found a place in the text-books on anatomy. As you see, the duodenal bulb is smooth walled and does not contract. Peristaltic waves in the duodenum begin not at the pylorus, but at a point 2 or 3 cm. beyond the pylorus. The second and third portions of the duodenum present a serrated shadow because of the Kerkringian folds. In this case food does not leave the stomach at the completion of every peristaltic contraction, but only at irregular intervals. Twice now we have seen the passage of a bolus into the duodenum with an interval of only two waves, but we may watch again for ten or twelve waves before any more passes out. In cases of achylia one sees food leaving the stomach at almost every wave, so that within a few moments the entire upper small intestine is densely filled. This same rapid emptying occurs in cases of adhesions in the region of the gall-bladder or when there are adhesions to the pancreas, without any actual mechanical obstruction.

As the patient stands here I am able to slip my gloved hand up behind the screen and by manipulation assist in the filling of the duodenal bulb. When one finds a normal duodenal bulb a negative opinion as to ulcer is warranted; hence in this case we may say that there is no roentgen evidence of duodenal ulcer. With the palpating hand we are also able to show with greater distinctness the lesser curvature and the greater curvature, and to push the stomach about so that we can see it under as many different conditions as possible; and in every respect the stomach appears to be normal. In order to make the study complete, however, we will also examine this patient in the horizontal position, so we will ask her to step out of the upright fluoroscope and to lie down on the horizontal fluoroscope. She may lie flat upon the back for a few moments while we change the switches and adjust ourselves ready for observation at the table.

In the horizontal fluoroscope the tube is underneath the table. We could have the screen suspended horizontally above the patient, but it is better to have it held by some one. Whoever holds it must take pains to see that the screen always blocks off the cone of *x*-light so that no *x*-rays will escape into the room, and I will take pains to keep the diaphragm opening reduced to the

smallest possible dimensions which will still permit us to see what is necessary.

Turning on the *x*-ray again, we observe that the stomach now assumes a shape very different from that exhibited in the upright

Fig. 39.—The normal stomach, patient standing.

position (Figs. 39 and 40). The opaque meal gravitates to the lowest point of the stomach, which now happens to be the fundus, so we are able to see the fundus very well, and to note the splenic notch, which here is normal. When the spleen is enlarged the splenic notch is correspondingly enlarged. I have seen the spleen

so large that the fundus of the stomach was on the right side, with the pylorus facing toward the left. There is a little material in the pyloric end of the stomach and scattered boluses throughout the small bowel, but the central portion of the abdomen, where we would expect to see the stomach shadow, is occupied instead by a gaseous brightening. This is due to the fact that, with the patient

Fig. 40.—The normal stomach, patient supine.

supine, the *magenblase* is not located high up under the left diaphragm in the fundus, but in the epigastrium, where the highest point of the stomach is now to be found. The stomach saddlebags over the spine. As the patient takes a deep breath, pushing out the abdomen at the same time, the stomach fills out more satisfactorily, and in some cases sufficiently so that we can make

out observations without manipulation. In the majority of cases, however, it is necessary to ask the patient to turn upon the right side for a few moments, at the same time placing the ulnar side of the left hand upon the epigastrium in such a manner that when the supine position is resumed the pressure of this hand will maintain the filling of the pyloric portion so that it can be studied. By a little gentle manipulation one is able to force considerable material through the pylorus into the duodenum, and at the same time, by making pressure with the little finger upon the duodenojejunal junction, compressing it between the finger and the spine, block off the duodenum. We are thus able to visualize the entire duodenum from pylorus to duodenojejunal junction. If we keep it thus obstructed, we see waves alternating in the onward and reverse directions, and can assure ourselves that the contour of this portion of the duodenum is normal. Thanks to this maneuver I have been able in a number of cases to diagnose, in addition to ulcer and gall-bladder adhesions, quite a number of cases of diverticula of the duodenum, dilatations of the ampulla of Vater, enlargements of the head of the pancreas, and, in 2 cases, tumors of the ampulla of Vater.

We will now dismiss the patient and, turning on the light, discuss any questions which may arise. We have spent considerably more time in this examination than one ordinarily requires, in order to demonstrate to you the technic of this study. Under ordinary circumstances we would see this patient again in two hours, and again at the end of five or six hours, in order to test the motility of the stomach, but in this particular case we have already answered the question asked us by the internist as to the *x*-ray evidence of duodenal ulcer.

QUESTION: This patient's history seems to be very suggestive. How do you account for the symptoms which she mentions?

DR. CASE: The stomach has been tritely called the "alarm-box of the abdomen." In fact, the statement has been made by a very famous surgeon that 95 per cent. of patients presenting gastric symptoms have no organic gastric disease, the symptoms arising from some other source. I know this is relatively so, for I have seen several patients whose cases were positively diag-

nosed duodenal ulcer from a clinical standpoint, where neither at operation nor at postmortem was there demonstrable any sign of an ulcer in the stomach or duodenum. Numerous investigators have called our attention to the fact that patients suffering from chronic colitis may present any of the right upper quadrant symptoms which suggest duodenal ulcer.

QUESTION: How about making plates in this case?

DR. CASE: In this particular instance the viscera have been seen very satisfactorily. There is no reason that I can see for spending the time and money in making plates. Dr. Moir has already made several plates of the gall-bladder region, in none of which are we able to recognize any gall-stones. We have seen the stomach and duodenum very satisfactorily. If we had not seen them satisfactorily, then I would by all means have ordered a number of plates. There are numerous devices for this purpose, all modifications of the so-called serial method, popularized by Cole, although practically in use by roentgenologists since the beginning of bismuth work. Instruments are now available whereby, within a few minutes, one may make multiple exposures of the pyloric end of the stomach upon one plate; for instance, four exposures upon an 8 x 10 inch plate, sixteen exposures upon a 14 x 7 inch plate (Fig. 41). Roentgenography is by no means a satisfactory substitute for fluoroscopy. In favorable subjects, which means 8 or 9 out of 10, where the fluoroscopic image is clear, the screen study of the contour of the gastric silhouette is very satisfactory. For absolute safety from criticism I usually make a few roentgenograms as a matter of record, but in this particular case I am not making any because I feel very certain there is nothing to be gained. Serial plates will, without fluoroscopy, give a good deal of information one needs to know, and in a large number of cases will, if the method be pursued thoroughly enough, permit a correct diagnosis. If one wishes a pictorial record of the case the roentgenograms are necessary.

Recognizing that in a large proportion of our cases presenting digestive complaints we will not find a gastric lesion, I feel that it would be ideal if every case could be examined from mouth to rectum. In fact, whenever it is possible, I make it a rule to ex-

amine the patient for three and one-half consecutive days, following the meal through from start to finish, and then re-examining the colon by means of the opaque enema. I have described this

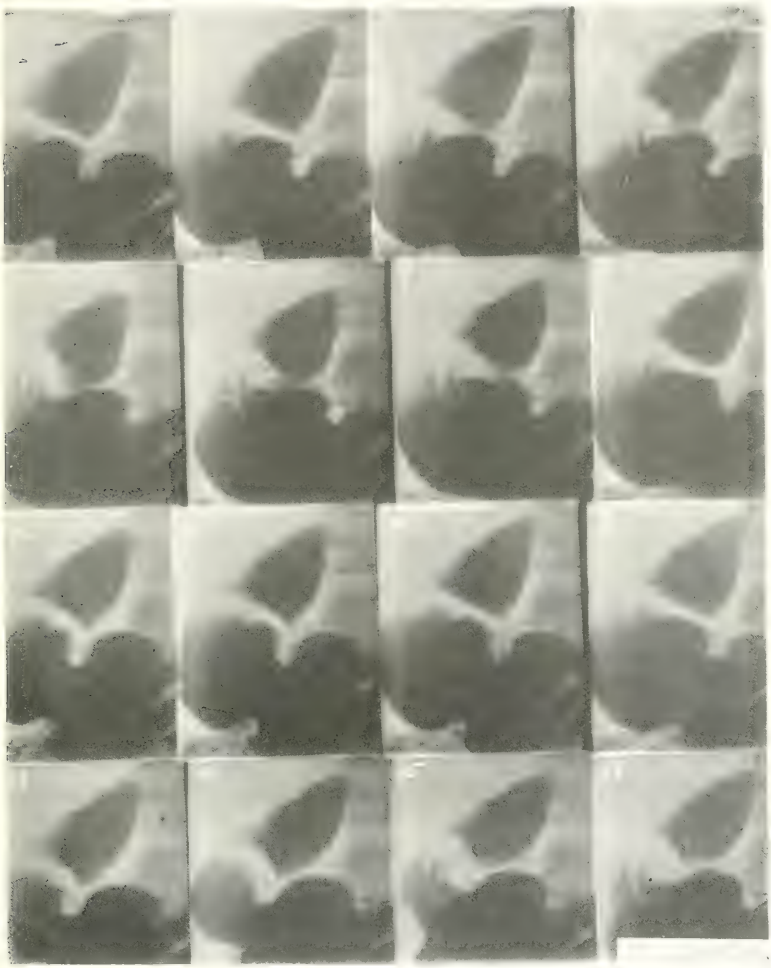


Fig. 41.—A print from a 14 by 17 inch plate containing sixteen serial exposures of the pyloric and duodenal regions. This method is complementary to, but not an adequate substitute for, fluoroscopy.

plan of examination in a previous lecture. After the meal I would see the patient at two and one-half hours, four and one-half hours, nine and one-half hours, twenty-six hours, and fifty

hours, ordering preparation for the opaque enema to be given somewhere near the seventy-fifth hour. Of course, in private practice one could shorten this somewhat, but for routine institutional work these hours have proved to be very satisfactory.

QUESTION: Can this work be done by one not a physician?

DR. CASE: Can satisfactory inspection, percussion, and palpation of the chest be done by one not a physician? The technician or trained nurse might just as reasonably expect to carry out an accurate physical examination of the patient by touch, percussing finger, or stethoscope as to properly interpret the *x*-ray findings, especially fluoroscopic *x*-ray findings. The most important part of *x*-ray work is not in the production of its image. One can hire a technician to install and start the apparatus, and to keep the current running, but the real practice of roentgenography, which means the interpretation of the roentgen findings, must be limited to physicians who have had a special training in *x*-ray work. Moreover, they must be physicians who have had at least a certain amount of clinical experience, as intimated earlier in this hour. The most important factor in estimating the value of an *x*-ray examination is the factor of the *x*-ray man himself.

QUESTION: What about the double meal and the six-hour residue?

DR. CASE: The double meal was devised by Haudek, of Vienna, to save time. It was not convenient for him to visit the hospital until nearly noon, so he had the patients given, in the early morning, beginning at 6 o'clock, an ordinary Rieder bismuth meal (2 ounces of bismuth salt in 10 or 12 ounces of farina mush or some other cereal). He began his work at the clinic at about 12 o'clock, so that these patients would come to him at the end of just about six hours after the meal. He would note the residue, if any, in the stomach, with special reference to position, shape, question of filling defects, etc. If there was no residue, he would note the advance which the meal had made in the bowel. The patient was then given another bismuth meal, very much after the manner we have described here today. The six-hour residue after a carbohydrate meal given in the manner described above has great significance. It is rarely observed in a stomach free from organic lesion. This question will be taken up at a future hour.

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